Amplia THERAPEUTICS

TARGETING PANCREATIC CANCER AND BEYOND

March 2026

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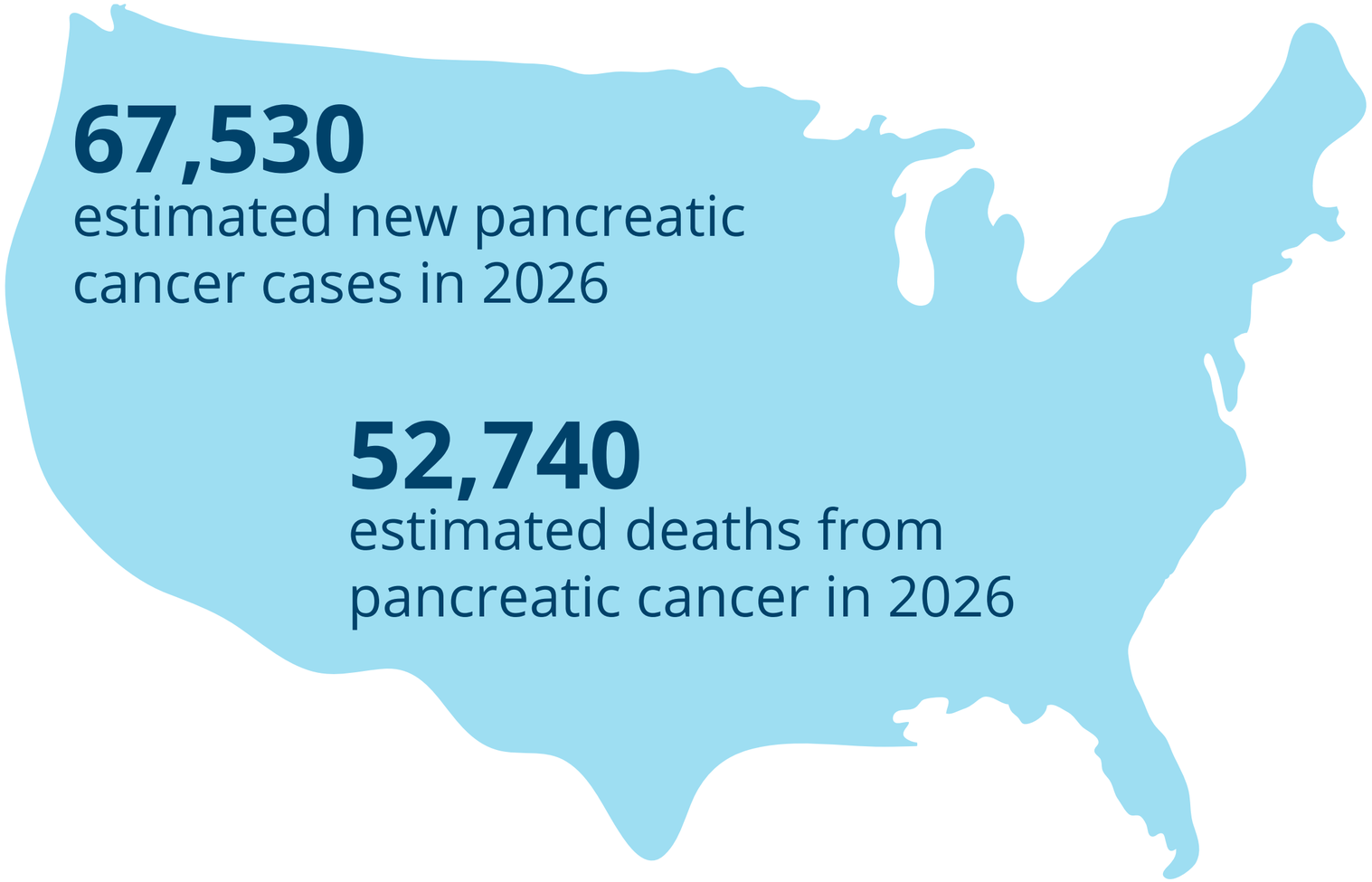
PANCREATIC CANCER

A devastating disease with limited treatment options



PANCREATIC CANCER

3rd

most common cause cancer death

3%

5 year survival rate for advanced disease

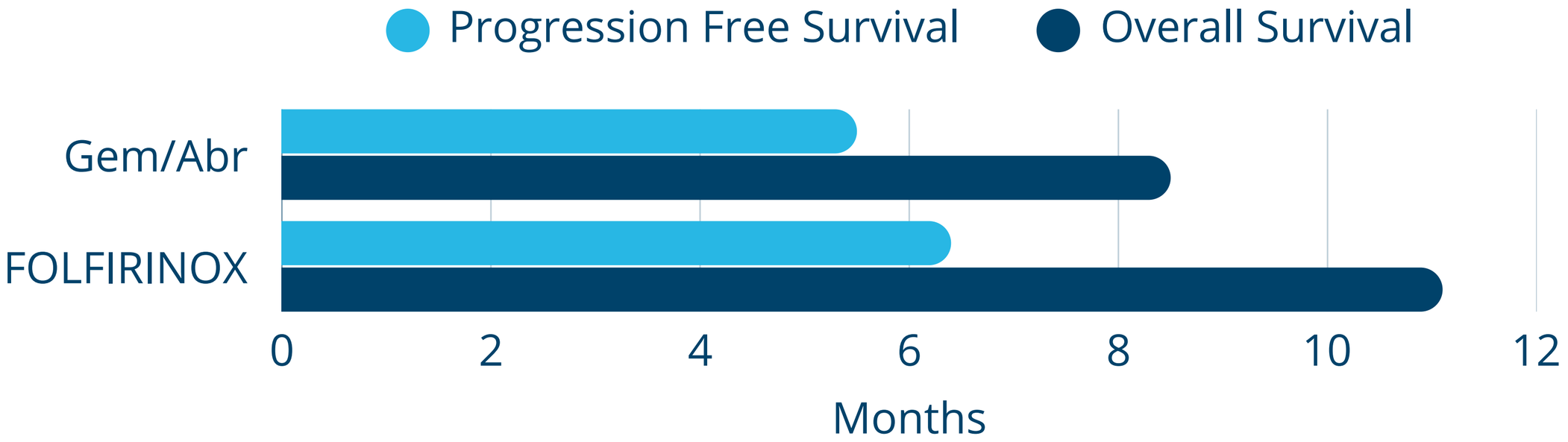
$3.8b

Global market predicted to grow to $14.4b by 2034

ADVANCED PANCREATIC CANCER

Approved treatments and outcomes

>50% patients diagnosed with metastatic disease



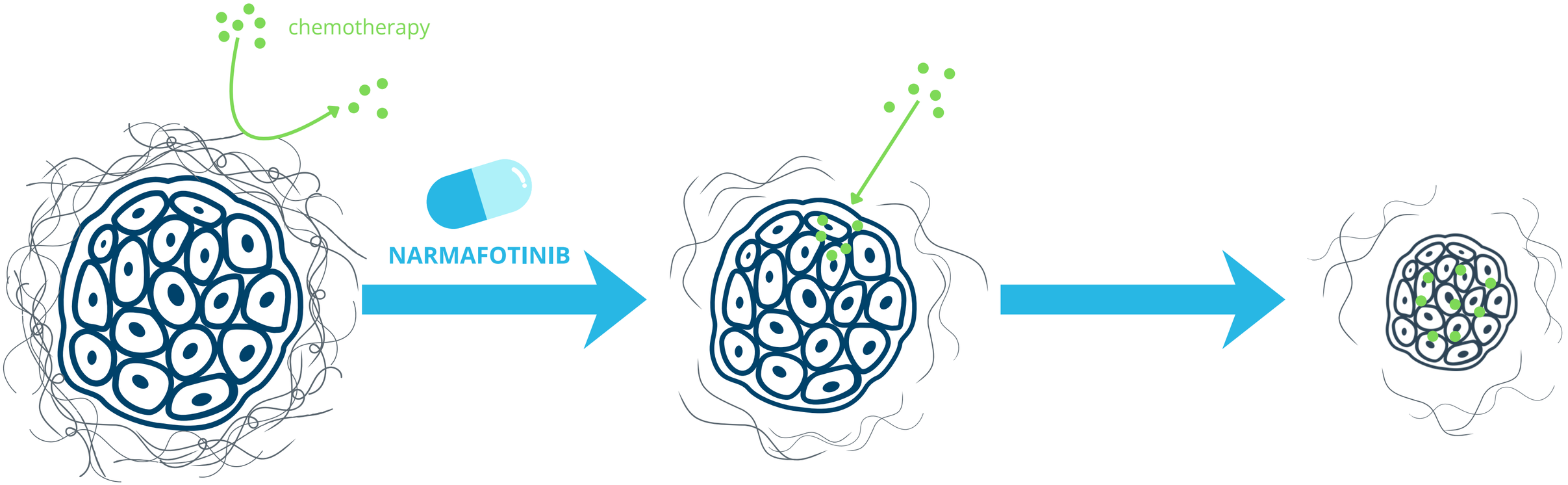
NARMAFOTINIB

Amplia’s lead drug is **ideally suited** for solid tumors like **pancreatic cancer**

1. Breaks down the **scar tissue** around the tumor
2. Blocks processes within cancer that **support cancer growth** *even in presence of chemotherapy*

BREAKING DOWN FIBROTIC SHIELD

Amplifying existing cancer treatments by reducing the fibrotic barrier that makes cancers difficult to treat

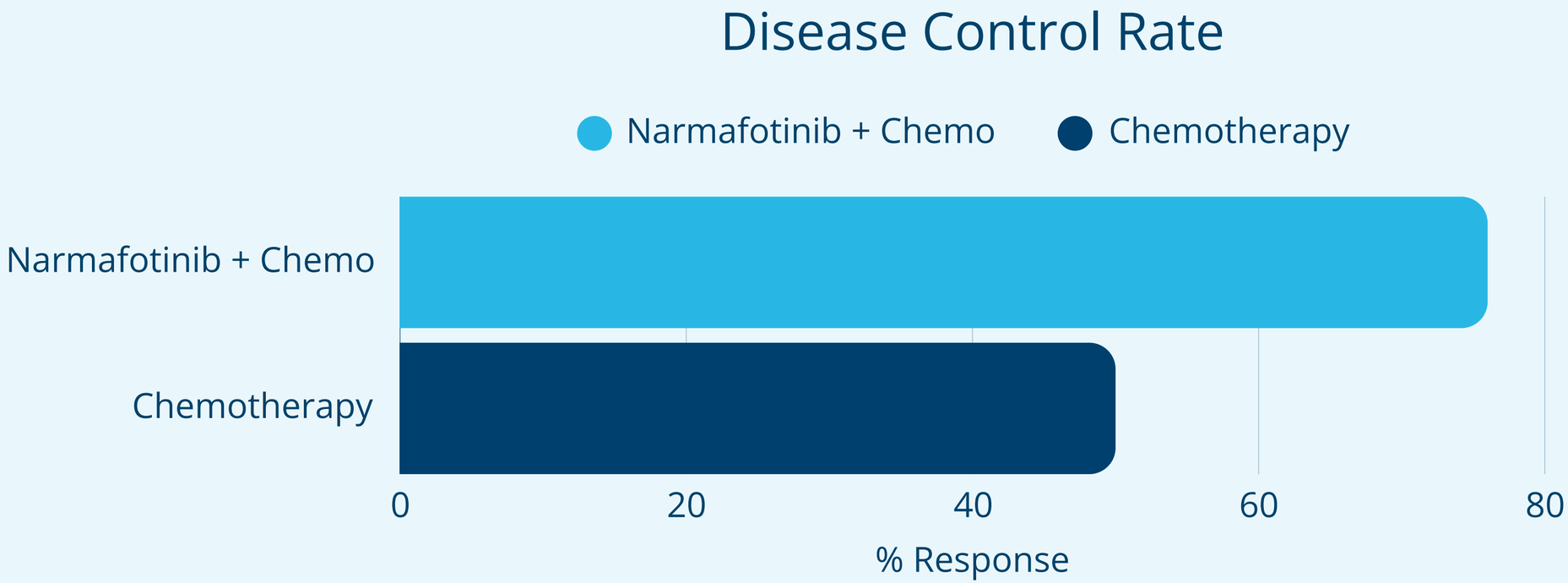


Cancer Associated Fibroblasts produce a dense fibrous matrix that surrounds the tumour and creates a barrier for chemotherapy

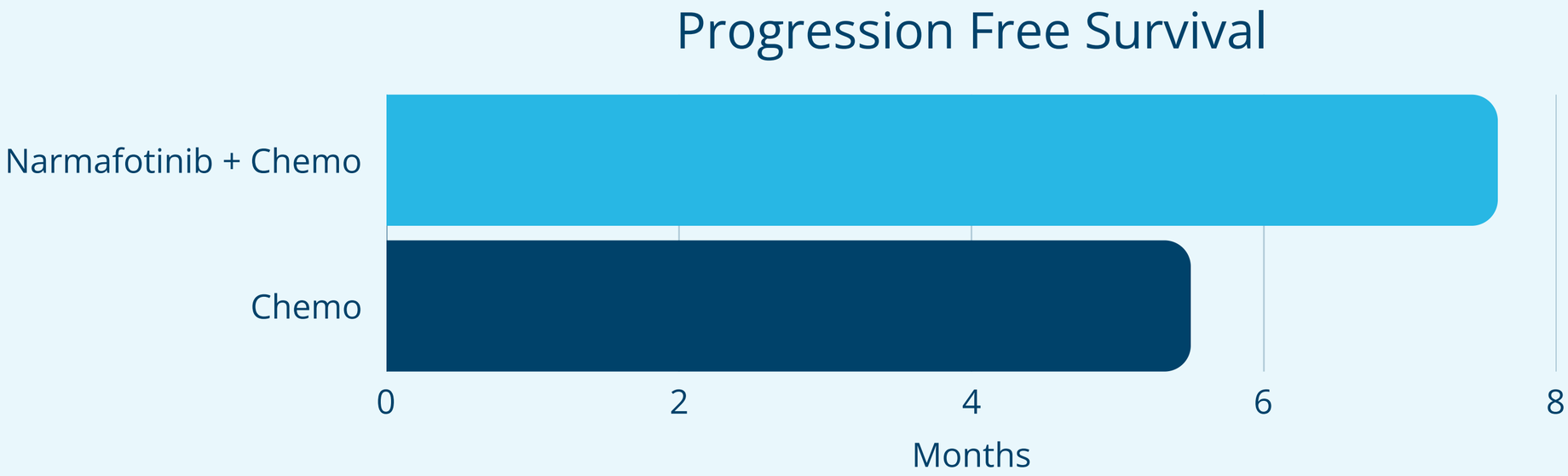
Narmafotinib acts to reduce the fibrous tissue thus allowing chemotherapy and other treatments to penetrate

Both narmafotinib and the chemotherapy can act directly on the cancer cells to kill them

ACCENT PANCREATIC CANCER TRIAL



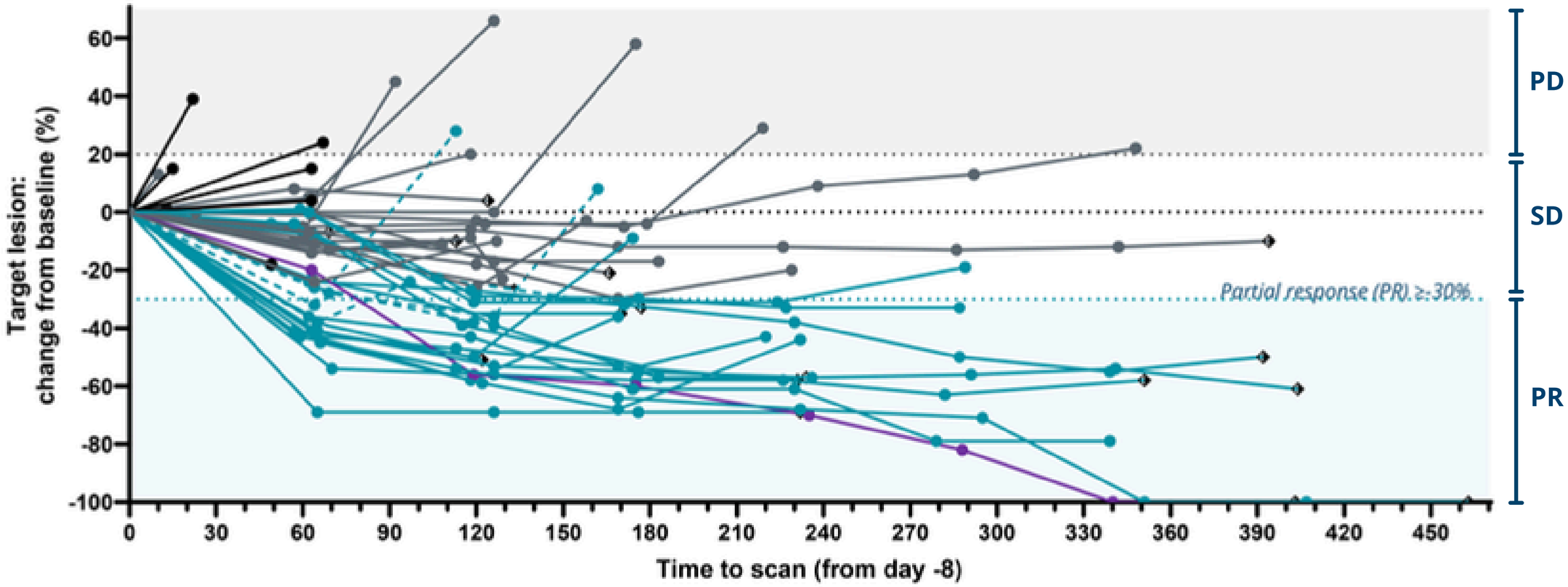
ACCENT PANCREATIC CANCER TRIAL



ACCENT TRIAL INTERIM DATA

Promising evidence of efficacy, durability and tolerability

Excellent durability observed



PETER'S STORY

An amazing outcome from a terrible diagnosis

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 trade'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 Jeanaali 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. 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 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 these 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 even. 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 2003 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 the 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 saw 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 reassessed 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 15 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 can get is important," 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 Zinet, a medical oncologist at the Jeanaali 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 Zinet 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 misere'. 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 this 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 trade 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."

NADIA KINAN

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

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

"I actually called the pathologist and said: Are you sure you're looking at the right specimen?"

PRASAD COORAY
ONCOLOGIST

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

CHRIS BURNS
AMPLIA CHIEF EXECUTIVE

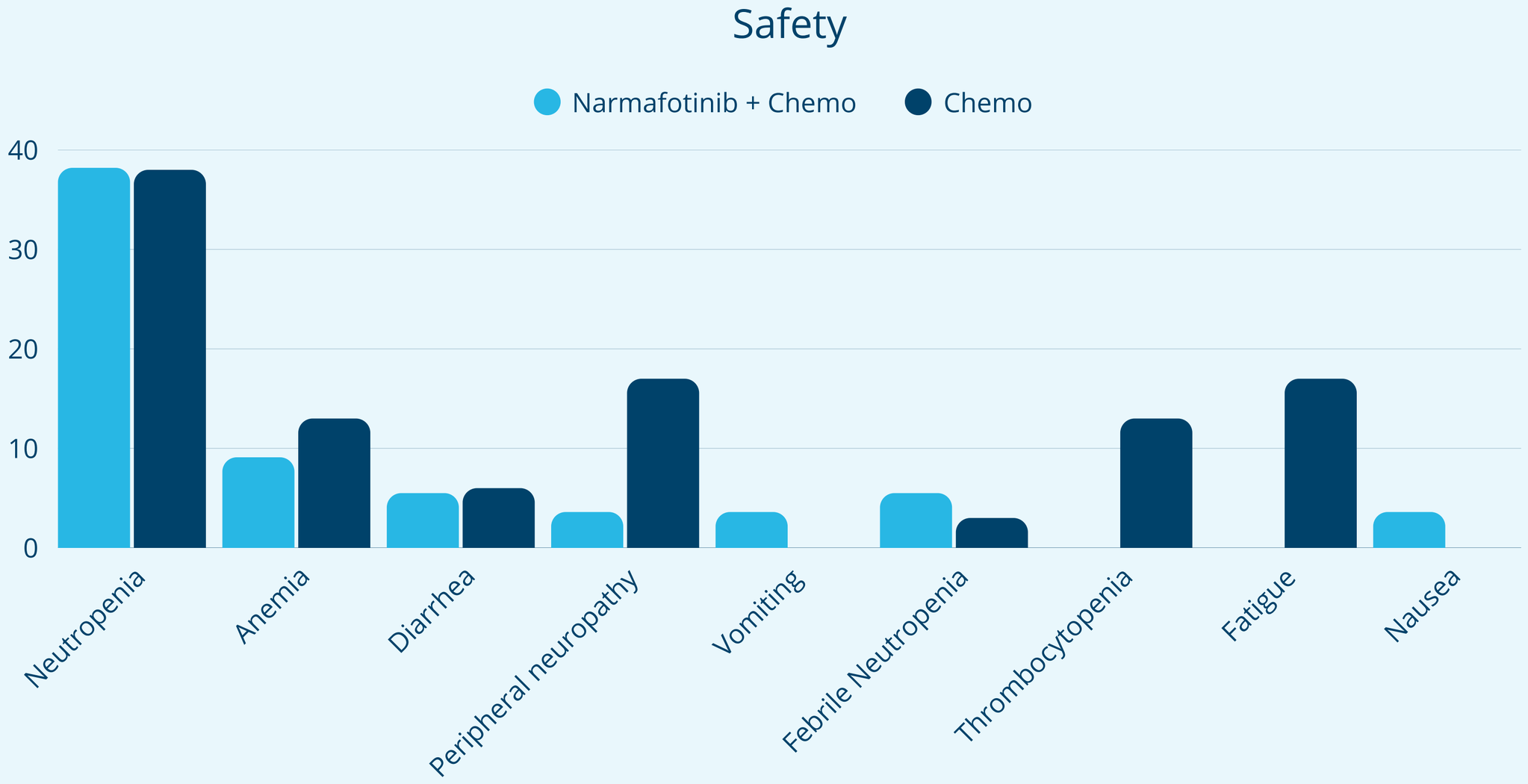
Confirmed Partial Response

Surgery to remove remaining tumour and metastases

No live tumour cells detected in removed tissue

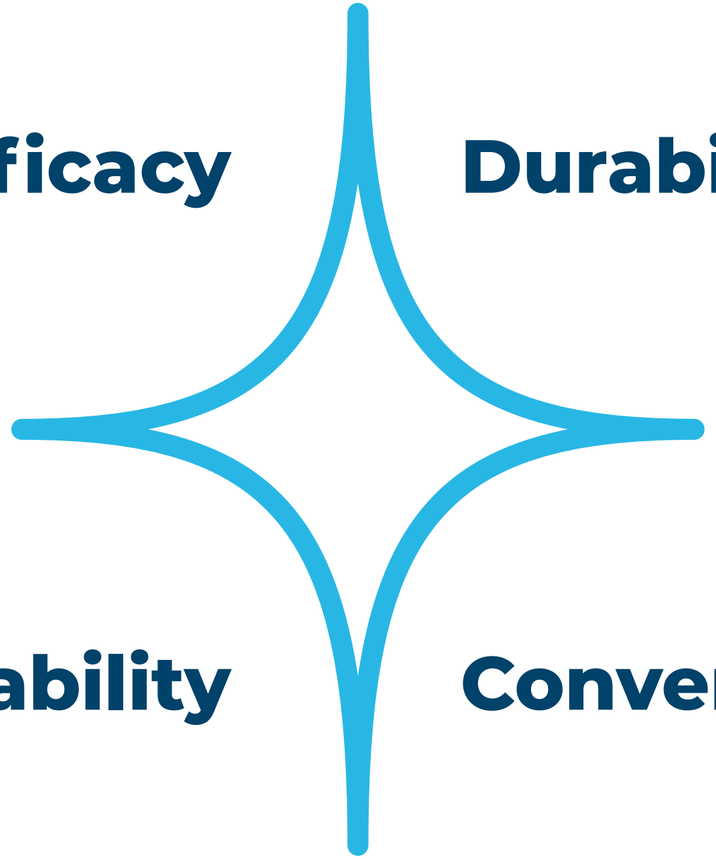
Pathological Complete Response

ACCENT PANCREATIC CANCER TRIAL



Compared to the MPACT Study (2013)

NARMAFOTINIB BENEFITS



CURRENT ACTIVITIES

Building on the promising data

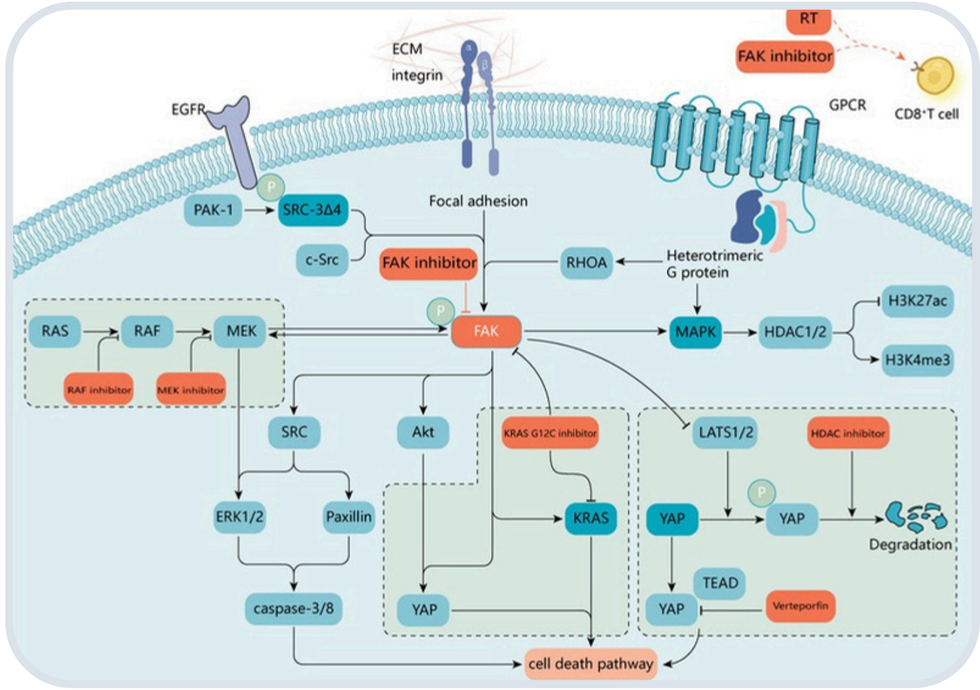
- **Complete ACCENT trial - *expect Q3 2026***
- **Plan for registration enabling trial - *begin end 2026***
- **Continue AMPLICITY trial - *US sites, pancreatic cancer***
- **Explore additional combination opportunities - *kRAS inhibitors***

NARMAFOTINIB AND KRASi

Strong rationale for amplified efficacy of kRASi

Growing evidence for clinical potential of FAK + kRAS inhibition

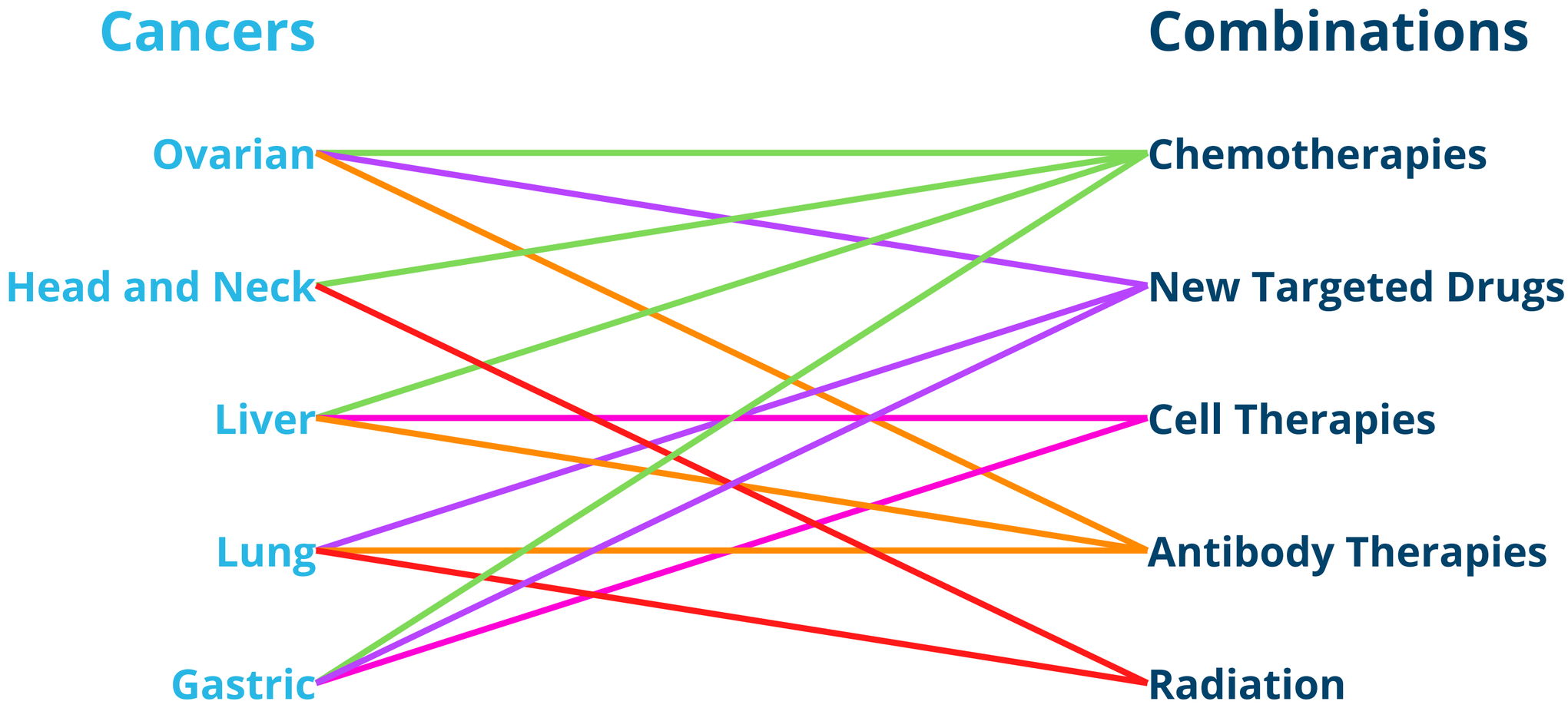
- Approval of defactinib + avutometinib in kRAS mutant LGSOC - VSTM
- Preclinical studies (Amplia, Inxmed) showing efficacy
- Promising early clinical data from Inxmed



Front. Cell Dev. Biol., 2022, 10

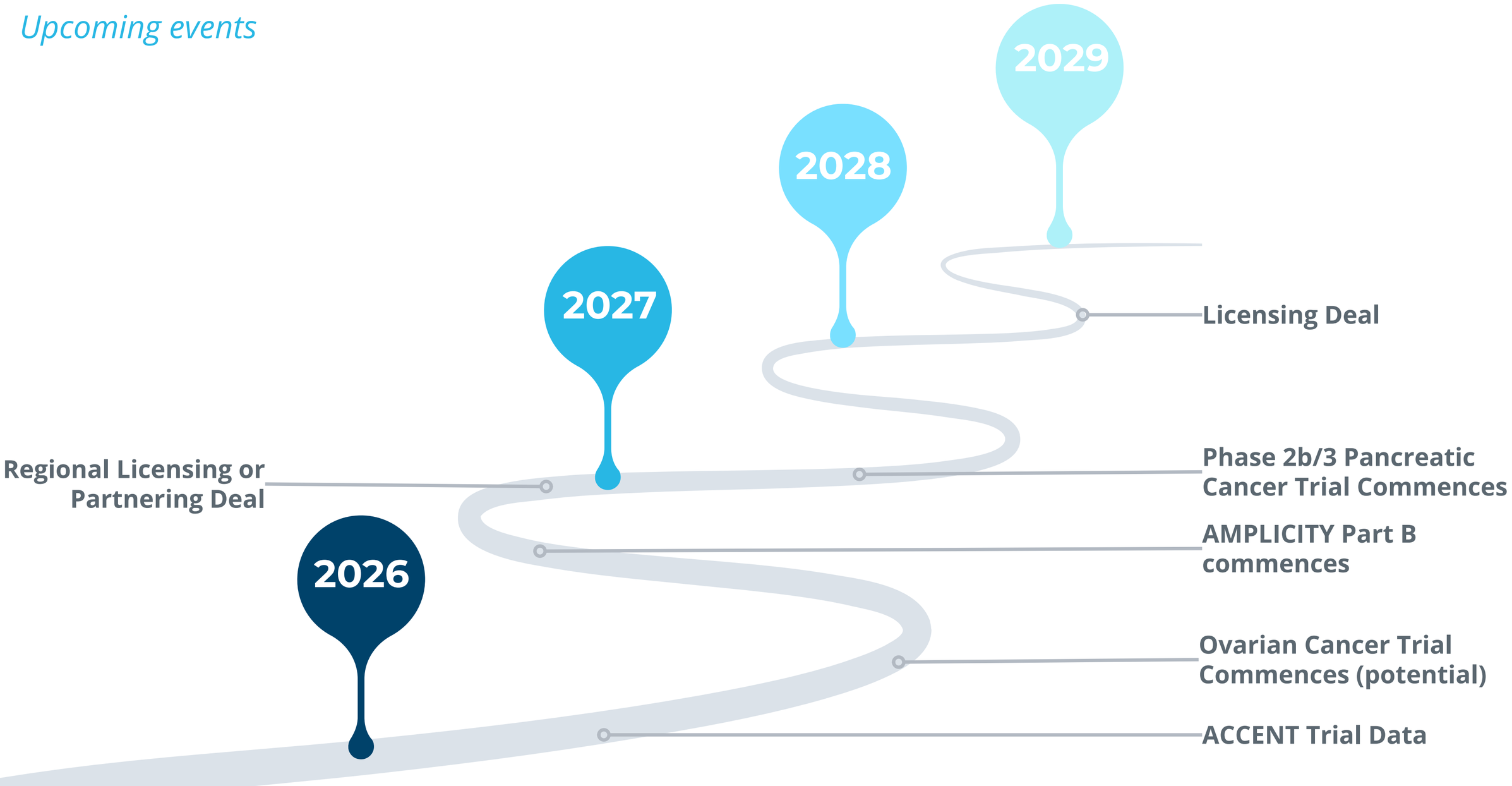
OPPORTUNITIES

Overcoming other difficult-to-treat cancers



KEY MILESTONES

Upcoming events



A RECORD OF SUCCESS

The team to progress this project

Developed drugs that have made it to market

Broad experience from leading pharma and biotech companies

Bristol Myers Squibb®

gsk

Pfizer

(osi) pharmaceuticals

cytopia
DEVELOPING BETTER DRUGS

receptos

mesoblast

zoetis

PolyActiva

Medicines Development for Global Health

Cancer Therapeutics CRC

Amplia
THERAPEUTICS

THANK YOU

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