16 SEPTEMBER 2026

ASX RELEASE

AMPLIA PRESENTS AT E&P HEALTHCARE CONFERENCE 2026

Melbourne, Australia: Amplia Therapeutics Limited (ASX:ATX; OTCQB:INNMF), is pleased to announce that CEO and Managing Director Dr Chris Burns will be presenting at the E&P Healthcare conference in Sydney, today.

A copy of the presentation is attached.

This ASX announcement was approved and authorised for release by the Managing Director 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 FAK, a protein over-expressed in multiple cancers and a drug target gaining increasing attention for its role in solid tumours. 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 being investigated in the ACCENT trial in advanced pancreatic cancer where it is dosed in combination with the chemotherapies gemcitabine and nab-paclitaxel (Abraxane®) in first-line patients. The trial has achieved its desired outcome in achieving a response rate of 36%, superior

to chemotherapy alone as reported for the benchmark MPACT study. A median Overall Survival (mOS) of 11.1 months has been reported along with an unprecedented complete response rate of 7.8%. A second trial (AMPLICITY) is being run at sites in Australia investigating the combination of narmafotinib with the chemotherapy mFOLFIRINOX in advanced pancreatic cancer patients, and a third trial in ovarian cancer (the PRROSE trial) is scheduled to begin recruiting later this year. A new trial, exploring the combination of narmafotinib with next-generation kRAS G12C inhibitor olomorasib in second-line NSCLC, is planned to start late 2026.

Amplia THERAPEUTICS

Targeting pancreatic cancer and beyond

SEPTEMBER 2026

ampliatx.com | @ampliatx

ASX: ATX | OTCQB: INNMF

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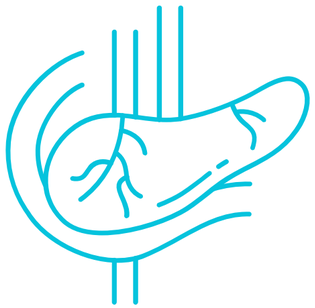
The FAK Opportunity

A large and unmet clinical need exists in cancers where FAK contributes to disease

Lead program

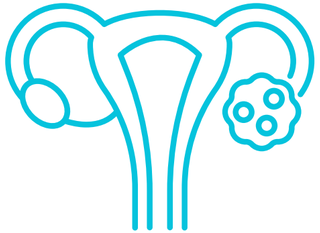
Pancreatic Cancer



US$2.5b market[1]

- Exciting data from ACCENT Trial
  Superior to chemo alone
- Phase 2b/3 registration enabling FDA Orphan drug & Fast track designation

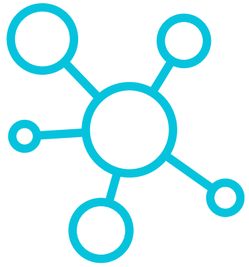
Ovarian Cancer



US$3.9b market[2]

- PRROSE Trial
  HGSOC patients | ANZGOG
- High FAK expression
  1 in 5 don't respond to chemo

kRAS Combinations



US$32b lung cancer market[3]
US$19b CRC market[4]

- Emerging opportunity
  80+ kRAS drugs in development
- Preclinical ATX models show narmafotinib can improve kRAS inhibitors

1. 2025 7MM's, Clarivate/ Nature Reviews Drug Discovery 25, 416-417 (2026); **2**. 2025 7MM's, Clarivate/ Nature Reviews Drug Discovery 25, 674-675 (2026); **3**. Global markets, Fortune Business Insights

Amplia THERAPEUTICS

Targeting fibrosis and resistance

Narmafotinib **breaks down fibrosis** surrounding the tumour, *and* **blocks** survival and **resistance pathways** in cancer cells

FAK drives 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

Chemotherapy **can now kill cancer** cells and **narmafotinib blocks resistance** processes

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ACCENT Trial in pancreatic cancer

Excellent tolerability and responses observed

Amplia THERAPEUTICS

ACCENT Pancreatic Cancer Trial

Phase 1b/2a clinical trial in Australia and South Korea

- **Narmafotinib plus standard-of-care chemotherapy**
- **Newly diagnosed patients**
- **Metastatic disease**
- **64 patients at top dose narmafotinib**

Narmafotinib

Taken orally in days preceding chemotherapy

Chemotherapy

Three infusions every four weeks

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ACCENT Pancreatic Cancer Trial

Narmafotinib + gemcitabine / Abraxane in first-line advanced pancreatic cancer

7.8%

Complete response rate

Superior to chemo alone (~0.2%)

5 CR's and 1 pCR from 64 pts

70%

Disease control rate

Chemo alone - 50%

8% CR, 28% PR, 34% SD

11.1 months

Median overall survival

Chemo alone - 8.5 mo

mPFS 7.7 mos
v 5.5 mos (MPACT)

No Extra Burden

Safety & tolerability

Over chemo alone

Main narma TEAEs: nausea, vomiting, diarrhoea

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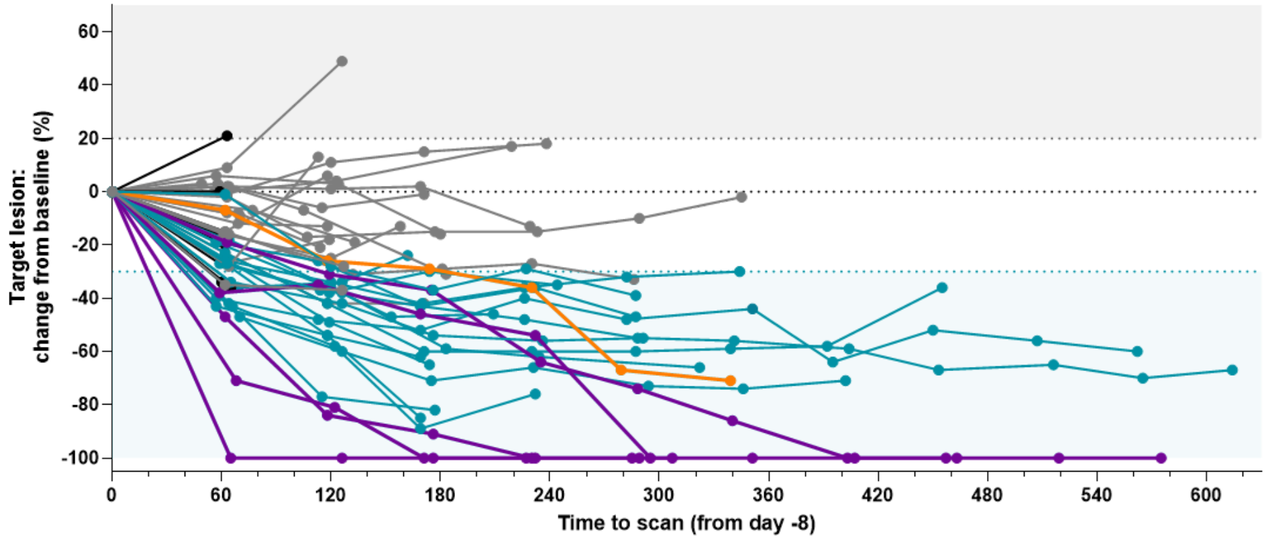
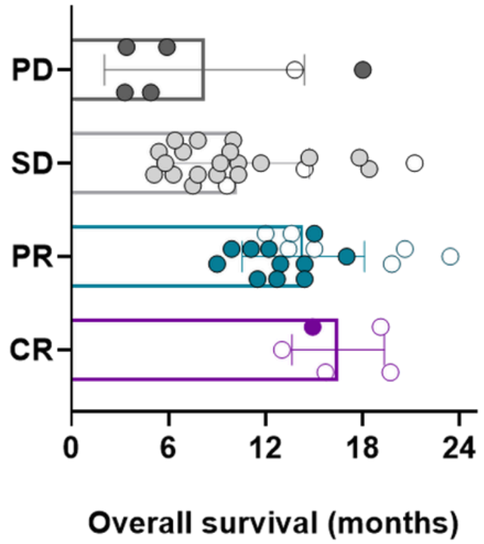
ACCENT Pancreatic Cancer Trial

Narmafotinib + gemcitabine / abraxane in first-line advanced pancreatic cancer

Deep and sustained tumour shrinkage



OS related to Response



White circles indicate patients alive at March review

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ACCENT registration pathway

A three-part registration pathway designed around FDA feedback

Underway

ACCENT Mini

Daily dosing at two dose levels

- Gemcitabine + Abraxane combination
- 12 patients - 2 cohorts
- 3–4 Australian sites
- Safety, tolerability, PK + efficacy
- Biomarker endpoints

Timeline

- **First patient dosing Sept 2026**
- Safety and PK review complete Q1 27

Final planning

ACCENT Reg - P2b

Builds on ACCENT Mini data to compare the two daily dosing levels head-to-head to **identify the optimal dose of narmafotinib** ahead of the Phase 3 study

- Following *Project Optimus** principles

Timeline

- FDA meeting planned Q2 27
- Start 2H 27

Final planning

ACCENT Reg - P3

Pivotal registrational study conducted with the dose selected from P2b

Designed to support FDA submission for narmafotinib approval in pancreatic cancer

* Project Optimus: https://www.fda.gov/about-fda/oncology-center-excellence/project-optimus

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The Pancreatic Cancer treatment landscape

New developments changing treatment journey

Chemotherapy predicted to remain a significant component of treatment paradigm

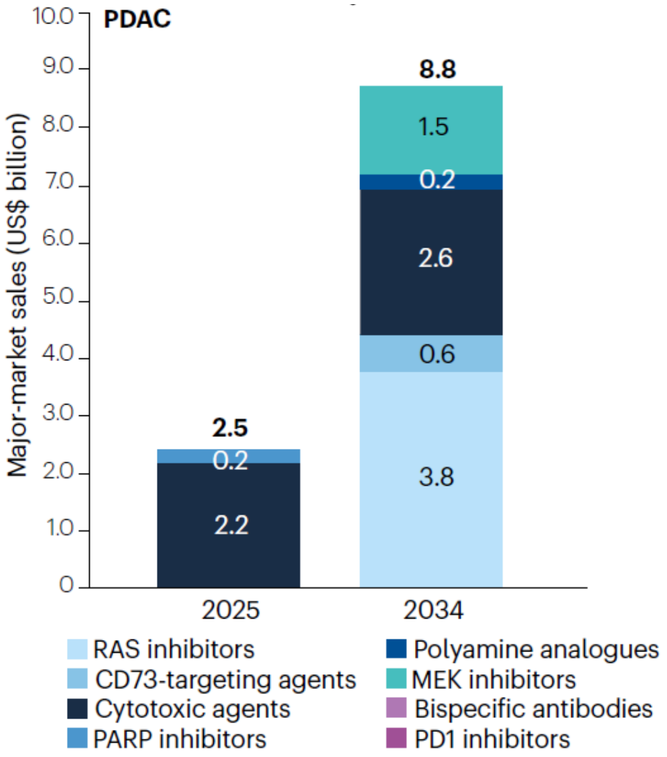
FOLFIRINOX and gemcitabine+Abraxane are main chemotherapy options

- Growing awareness that gemcitabine+Abraxane combination has similar efficacy with improved tolerability
  - US$6,500-7,500/month
- Recently approved NALIRIFOX (approved in US) offers good efficacy with reduced toxicity
    - US$13,610/month

kRAS/RAS inhibitors most exciting of new treatment options

- Daraxonrasib US approval for second-line advanced PDAC in August

Market Size (7 MM)



Nature Reviews Drug Discovery 2026, 25, 416-417 (Clarivate)

kRAS Inhibitors

An exciting new class of anti-cancer therapies - ideally suited for FAK inhibitor combination

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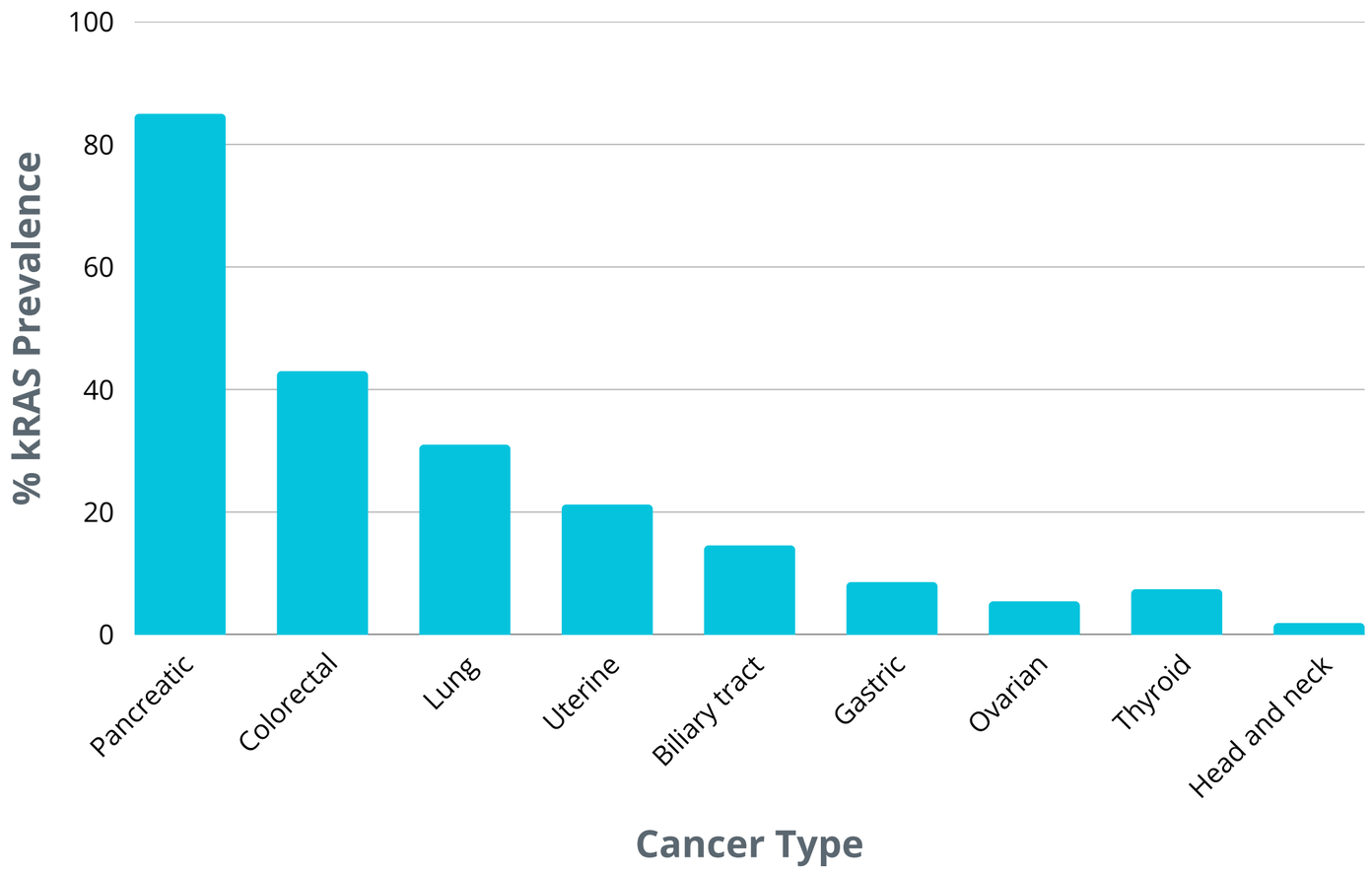
kRAS mutations prevalent in cancer

Significant cancer driver in multiple indications



Clinical studies demonstrating good activity

Resistance and tolerability significantly limits long term treatment

80+ kRAS drugs now in clinical development
Across pancreatic cancer, NSCLC, colorectal cancer and others

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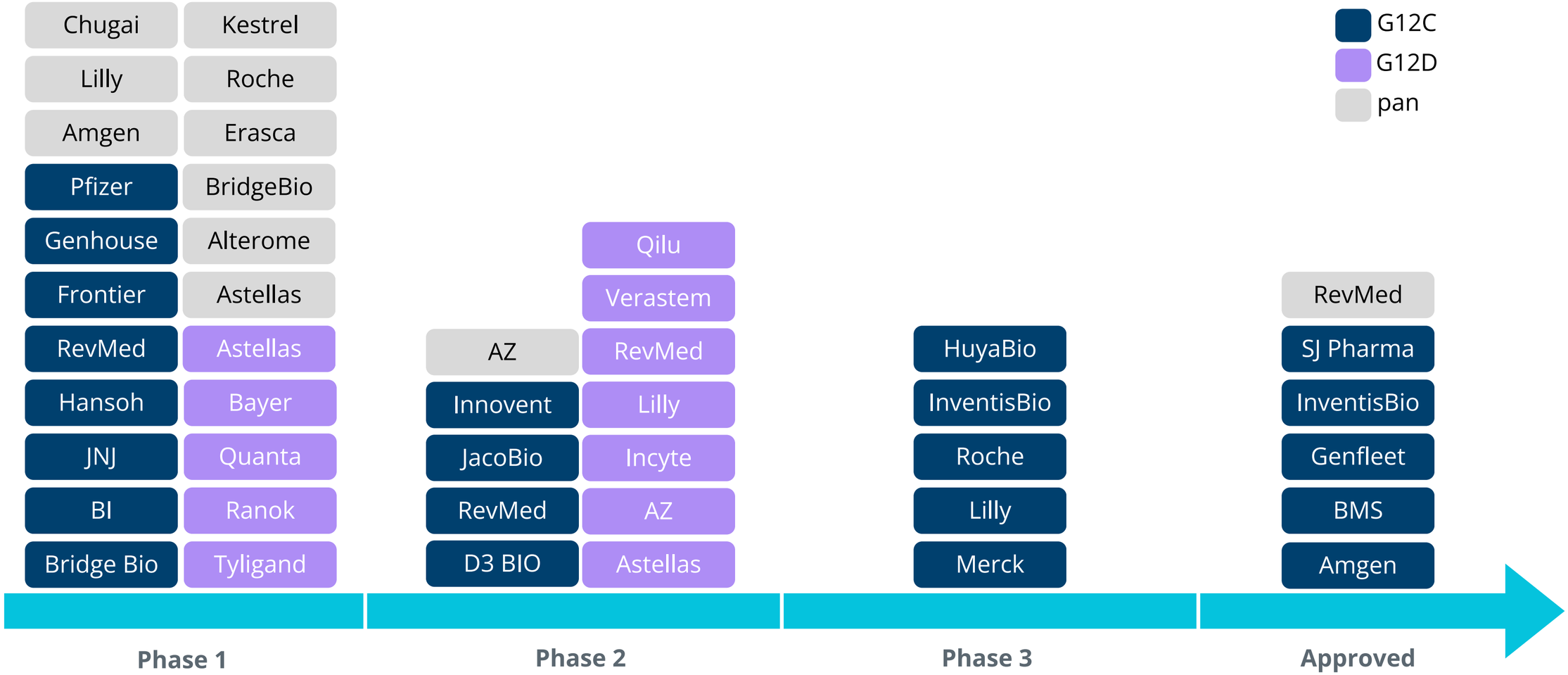
Significant competition in development

Combinations critical to development strategy



The Opportunity Ahead

Combinations critical to development strategy

'It is now generally accepted that the long-term effectiveness of KRAS inhibitors will not be achieved through mono-therapy, but rather through combination therapy.'

Trends in Cancer

CellPress OPEN ACCESS

Feature Review

KRAS inhibitors: resistance drivers and combinatorial strategies

Tamara Isermann[1,2], Christine Sers[1,2], Channing J. Der[1,3,4], and Bjoern Papke[1,2,3,4,*]

In 1982, the RAS genes *HRAS* and *KRAS* were discovered as the first human cancer genes, with *KRAS* later identified as one of the most frequently mutated oncogenes. Yet, it took nearly 40 years to develop clinically effective inhibitors for RAS-mutant cancers. The discovery in 2013 by Shokat and colleagues of a druggable pocket in KRAS paved the way to FDA approval of the first covalently binding $KRAS^{G12C}$ inhibitors, sotorasib and adagrasib, in 2021 and 2022, respectively. However, rather than marking the end of a successful assault on the Mount Everest of cancer research, this landmark only revealed new challenges in RAS drug discovery. In this review, we highlight the progress on defining resistance mechanisms and developing combination treatment strategies to improve patient responses to KRAS therapies.

Highlights

After nearly 40 years of effort to develop a clinically relevant KRAS inhibitor, Kevan Shokat's group identified the first mutant-selective inhibitor targeting $KRAS^{G12C}$. This breakthrough reignited interest in KRAS as a drug target, culminating in FDA approval of the first mutant-selective KRAS inhibitors.

Despite the initial favorable response to $KRAS^{G12C}$ inhibitors, resistance and disease progression are observed in most patients.

Numerous combinatorial therapies involving KRAS inhibitors are in clinical trials aiming to overcome therapy resistance. Additionally, other mutant-specific inhibitors (e.g., $KRAS^{G12D}$), as well as pan-KRAS inhibitors or RAS(ON) multiselective inhibitors, are entering clinical trials and demonstrating promising results.

With the growing number of RAS inhibitors, patients with mutated KRAS are edging closer to an effective targeted therapy.

The rise of RAS oncogenes as undruggable cancer targets

The year 1982 marked the beginning of a new era in cancer research, with the discovery of *HRAS* and *KRAS* as the first human cancer genes [1,2]. Since then, the RAS gene family, comprising *HRAS*, *NRAS*, and *KRAS*, has been identified as one of the most important oncogene groups in cancer [3,4]. Among all RAS genes, *KRAS* is the most frequently mutated, accounting for ~80% of all RAS mutations [5,6]. Notably, RAS mutations exhibit significant tissue specificity, with the most common and lethal cancer types, such as colorectal cancer (CRC), pancreatic ductal adenocarcinoma (PDAC), and non-small cell lung cancer (NSCLC), being prominently associated with *KRAS* mutations (43%, 85%, and 31%, respectively) (Figure 1A) [7]. By contrast, *HRAS* mutations are commonly linked to skin cancer (excluding melanoma) (6%), bladder cancer (5%), and head and neck cancer (7%), while *NRAS* mutations are prominent in melanoma (23%), thyroid cancer (11%), and leukemia (9%)' (Figure 1B). RAS proteins are key regulators of oncogenic signaling pathways, including mitogen-activated protein kinase (MAPK) and phosphoinositide 3-kinase (PI3K) pathways. This is further underscored by the high frequency of cancer-driving mutations in RAS downstream effectors (Box 1), including oncoproteins, such as BRAF (8%), AKT1/2 (4%), MEK1/2 (2%), and PIK3CA (13%)', which all result in constitutive protein activity promoting tumor growth and survival. The important role of RAS proteins and RAS downstream effectors as drivers of cancer has led to an intense search for effective RAS inhibitors.

Early on, experimental studies using cell culture and mouse models confirmed mutant RAS as a critical driver of cancer development and progression, further validating it as a favorable cancer target [8,9]. However, the tertiary structures of RAS proteins revealed the lack of deep hydrophobic pockets on the surface, limiting opportunities for the design of potent and selective small-molecule inhibitors [10,11]. In addition, the exceptionally high picomolar affinity of RAS for GTP, together with the high cellular concentration of GTP (~500 μM) renders RAS targeting significantly more challenging compared with kinases, which bind ATP with micromolar affinity and ATP concentrations in the millimolar range [12–14]. This contributed to the longstanding belief that RAS was undruggable [15,16]. Consequently, early strategies aimed at targeting RAS were primarily indirect.

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Trends in Cancer, February 2025, Vol. 11, No. 2

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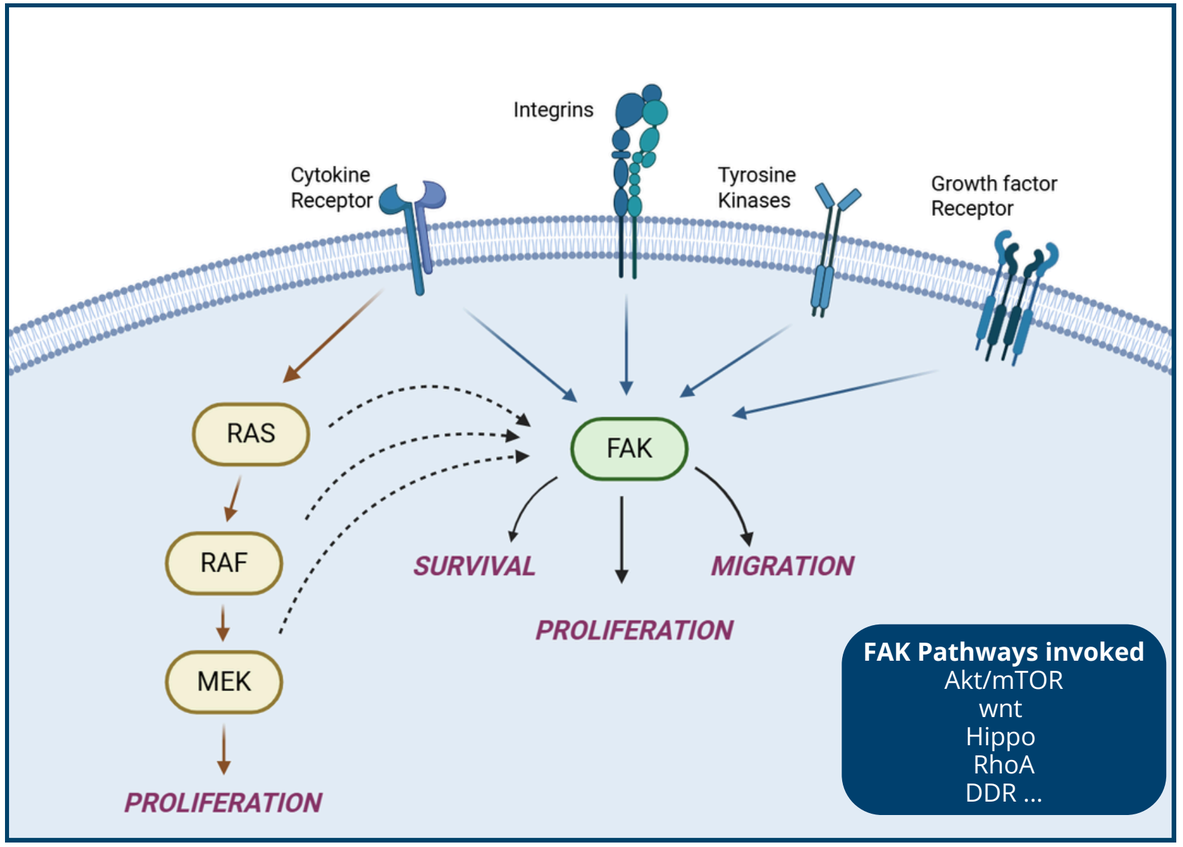
Narmafotinib and kRAS inhibition

Overcoming acquired resistance to kRAS inhibitors

FAK signaling is an escape mechanism to blockade of RAS pathway

Growing evidence for clinical benefit of FAK + kRAS inhibition

- Approval of defactinib + avutometinib (Co-Pack) in kRAS mutant LGSOC - *Verastem*
- Promising early clinical data from *Inxmed (Lancet Resp. Med.* 2026)
- Preclinical studies showing improved efficacy in combination



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Lilly Collaboration - Background

Enhancing efficacy for next-generation kRAS G12C inhibitor

FDA-approved G12C targeted drugs - first generation

- Adagrasib (BMS); Sotorasib (Amgen)
- Clinical data:
  - Promising efficacy signals resulted in accelerated approvals
  - Tolerability issues have limited market penetration

Olomorasib - a next-generation kRAS G12C inhibitor

- Improved potency and selectivity
- Promising P1/2 data has supported further development
- Currently undergoing two global Phase 3 studies in 1L NSCLC

Drug	Trial	Setting	mPFS	mOS	ORR
Adagrasib	KRYSTAL-12	Phase 3 2L+ NSCLC	5.5 mos	Immature	31.9%
Sotorasib	CodeBreaK 200	Phase 3 2L+ NSCLC	5.6 mos	10.6 mos	N/A
Olomorasib	LOXO-RAS-20001	Phase 1/2 2L NSCLC	8.1 mos	NR	41%

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Lilly Collaboration - Detail

Enhancing efficacy for next-generation kRAS G12C inhibitor

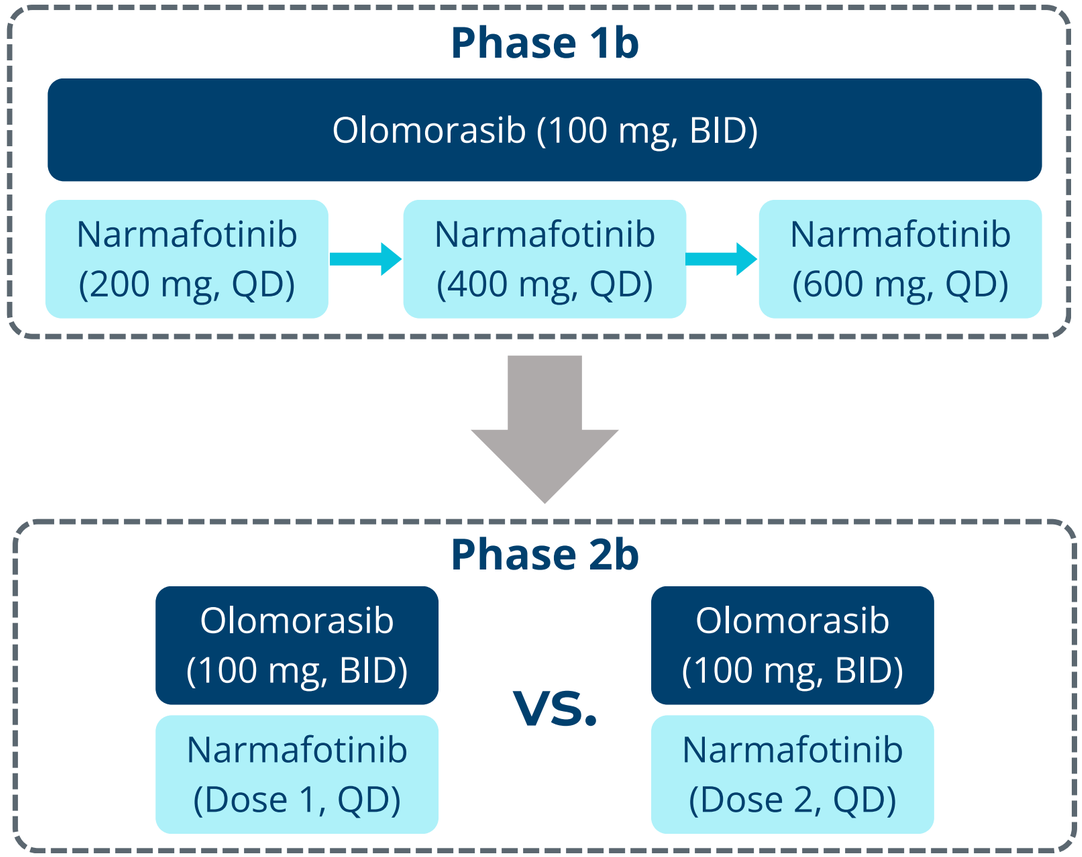
Previously treated KRAS G12C-mutant advanced or metastatic NSCLC

Phase 1b/2b study

- Dose escalation of narmafotinib with standard dose olomorasib (approx. 20 pts)
- Randomised 2-dose comparison – following Project Optimus guidelines (approx. 40 pts.)
- Australian and US sites

Contractual

- Amplia to sponsor and fund study out of existing funds
- Lilly to supply olomorasib in-kind for use in the study
- Resulting data to be shared between both parties
- Non-exclusive



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Lilly Collaboration

Important validation of the potential of our science

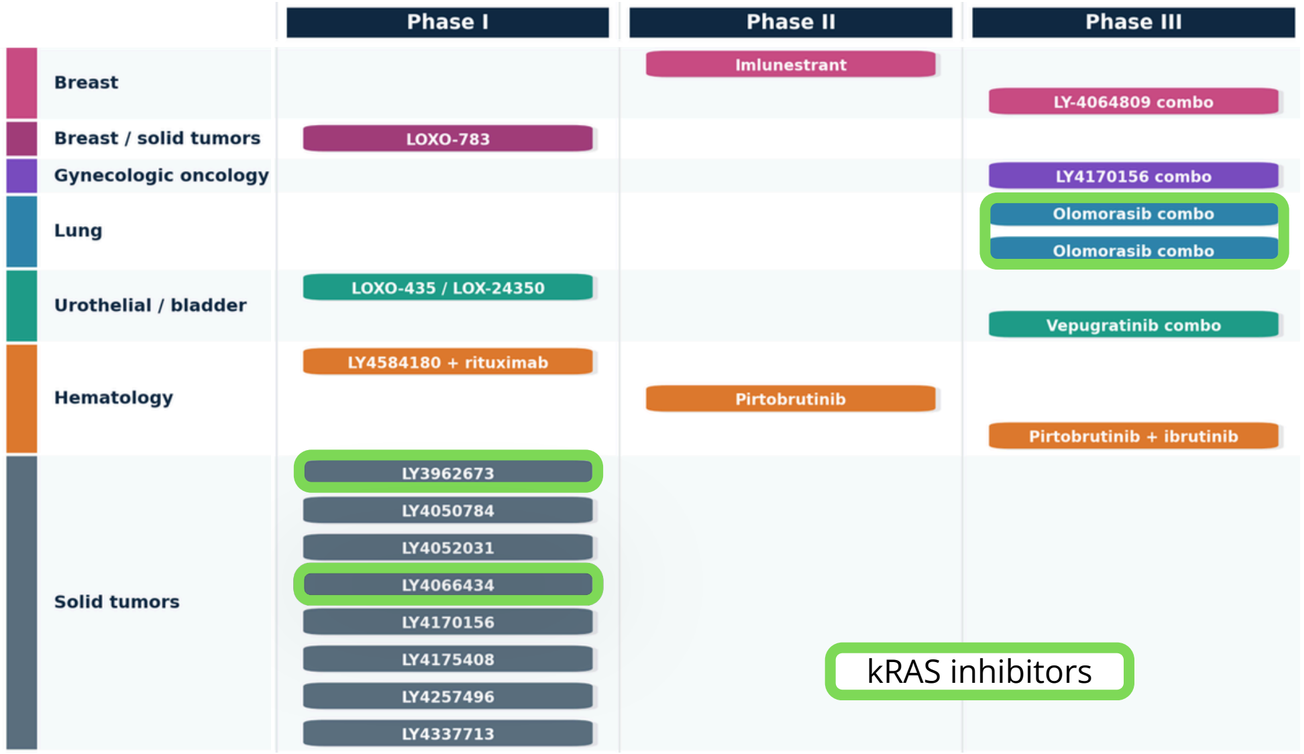
Eli Lilly and Co.

- Market Cap US$1.1 T

- Marketed oncology drugs
  - Verzenio (abemaciclib)
  - Jaypirca (pirtobrutinib)
  - Retevmo (selpercatinib)

- A deep pipeline of small molecules drugs, radiopharmaceuticals and biologicals
  - 3 kRAS inhibitors in development (G12C, G12D, and pan-kRAS)

Lilly Oncology Pipeline



PRROSE Trial in ovarian cancer

Building on promising preclinical and literature data

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Ovarian Cancer

70%

of patients are diagnosed at advanced stage where 5-year survival is 20%

20%

of patients will be resistant or develop resistance during initial treatment

~50%

of an ovarian tumour is fibrotic - a key driver of drug resistance

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PRROSE Trial

The study is an investigator-initiated clinical trial led by **Dr Gwo Yaw Ho of Monash Health** and Monash University, and sponsored and coordinated through **ANZGOG**

~20 Patients
HGSOC
poor chemo response

→

Treatment
Narmafotinib +
Carboplatin / paclitaxel

→

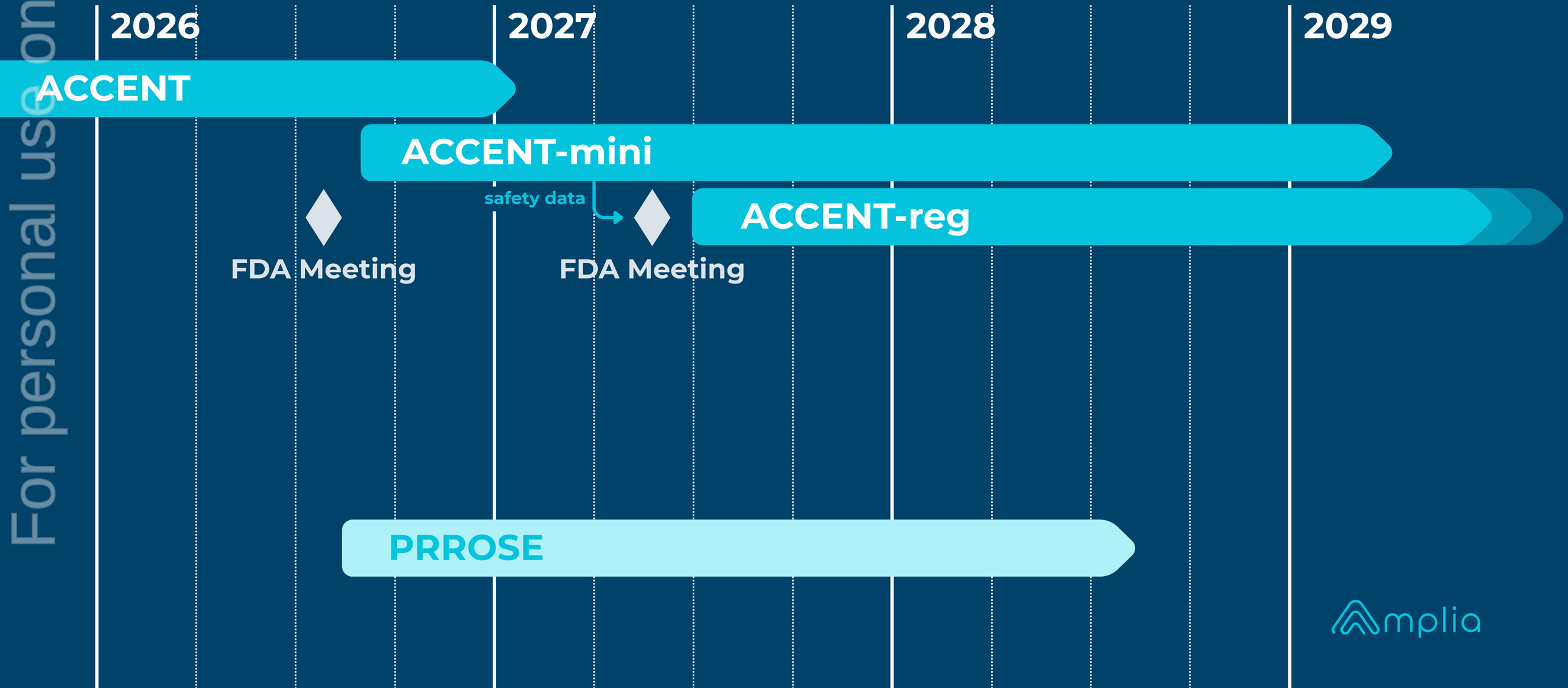
Surgery
interval debulking
surgical resection

→

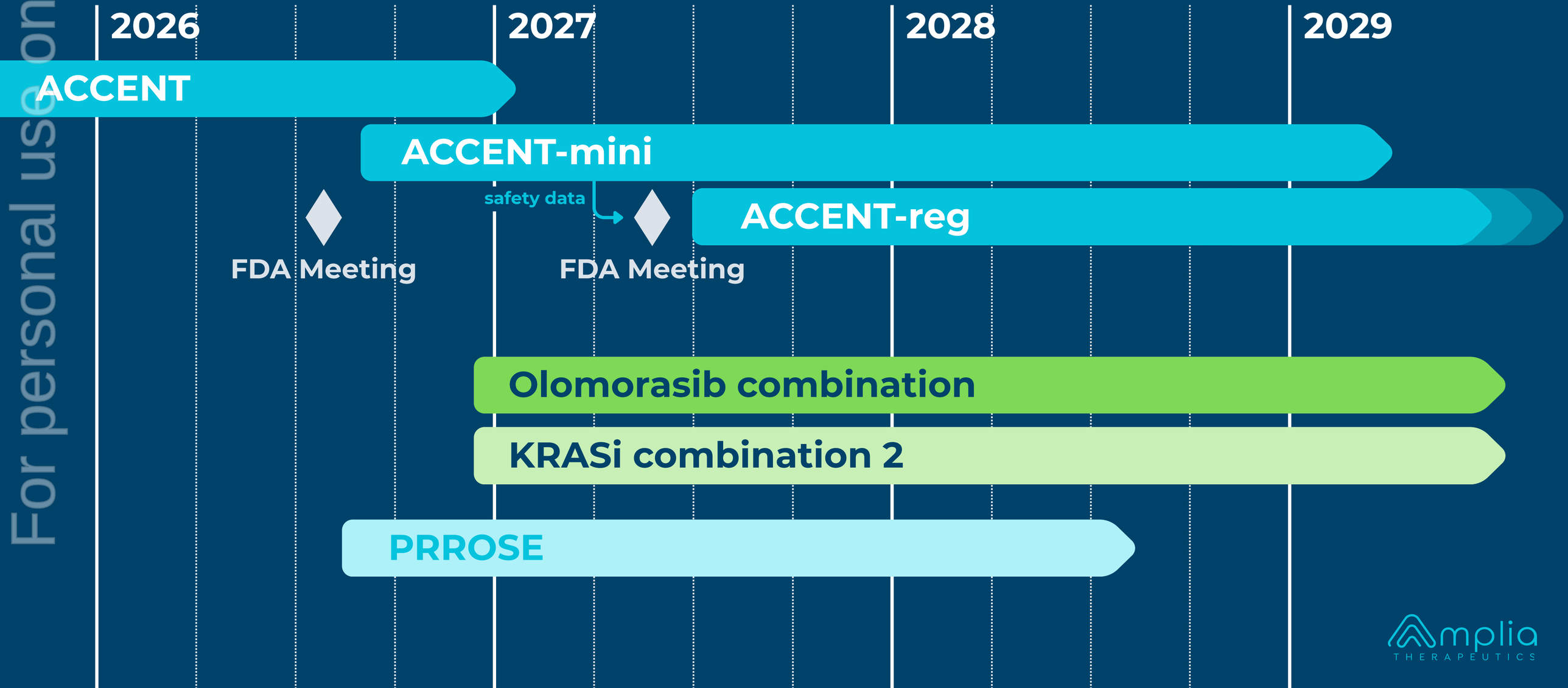
Endpoints
Safety, surgical eligibility
tissue + biomarkers

TRIAL PARTNER ANZGOG
Australia New Zealand Gynaecological Oncology Group — international cooperative trials network spanning major hospitals across Australia and New Zealand

Development Timeline



Development Timeline



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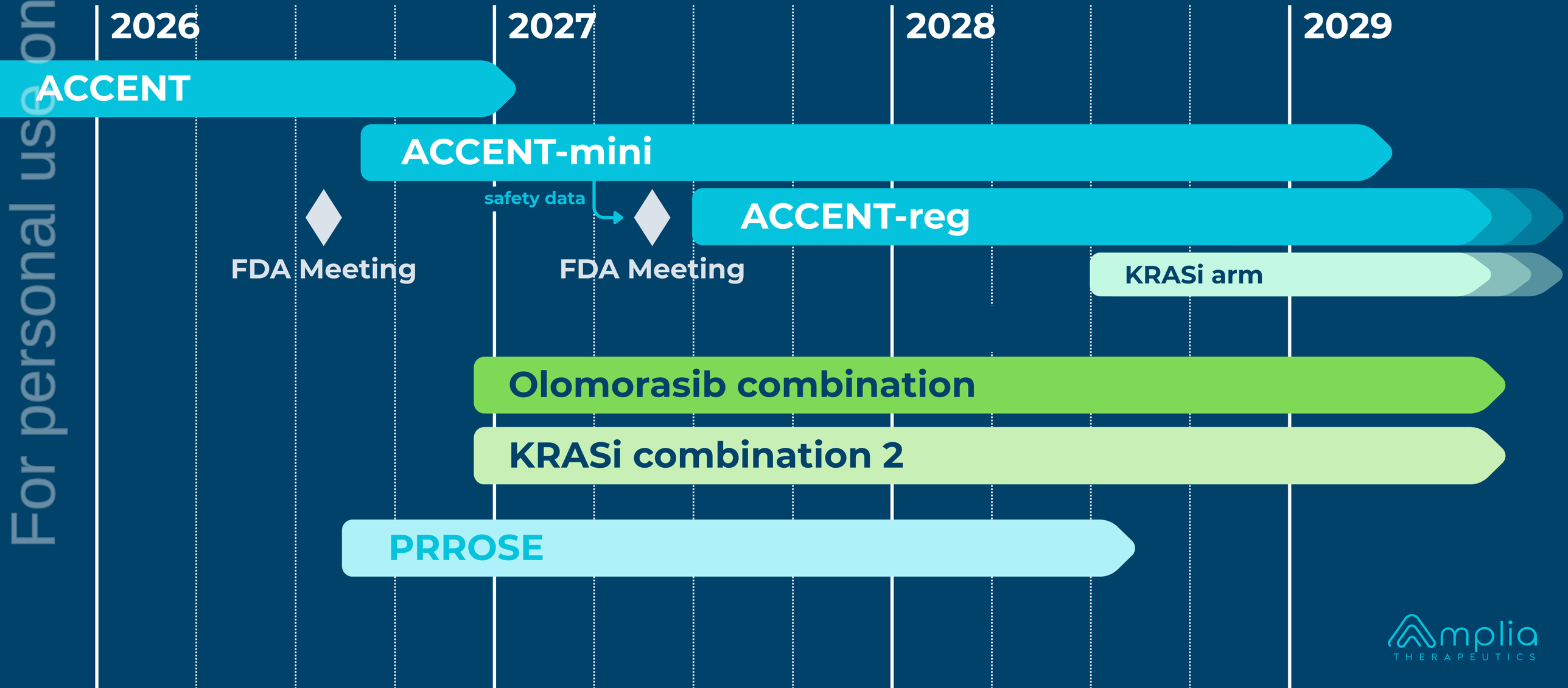
Development Timeline



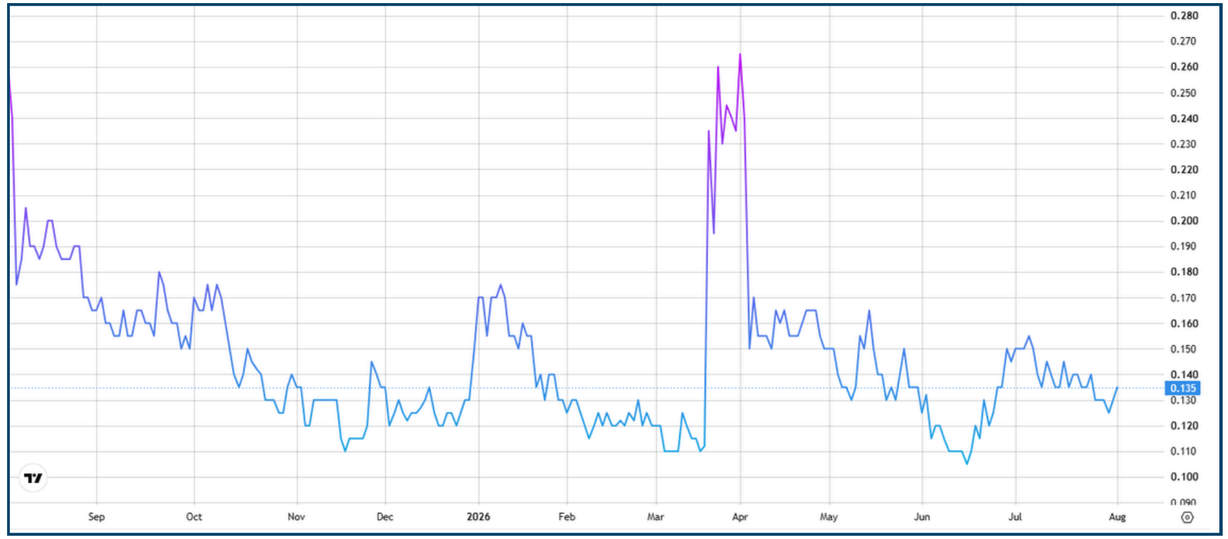
Amplia THERAPEUTICS

Corporate Summary

ASX:ATX | OTCQB:INNMF

Share price (15-Sep-2026)	A$0.12
Shares on issue	513.3M
Market cap (15-Sep-2026)	A$61.60M
Cash at hand (30-Jun-2026)	A$24.0M
Substantial Shareholders	• Platinum Investment Management Ltd • Acorn Capital Ltd • Blueflag Holdings Pty Ltd • Pengana Capital Group

12 month share price chart



Board and Management

World-class expertise

BOARD

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Jonathan Barlow

LLB BSc GDipBA GAICD

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Amplia THERAPEUTICS

Thank You

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