06 AUGUST 2026

ASX RELEASE

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 Queenstown, NZ today.

This ASX announcement was approved and authorised for release by the Managing Director of Amplia Therapeutics

Investor Contact:
Dr Chris Burns
Chief Executive Officer
chris@ampliatx.com

Media Contact:
H^CK Director, Haley Chartres
haley@hck.digital
+61 411 235 692

U.S. Contact:
Robert Giordano
rjgiordano@ggrouplifesciences.com
+1 917 327 3938

U.S. Media:
media@ampliatx.com

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

BIOSHARES 2026

ampliatx.com | @ampliatx

ASX: ATX | OTCQB: INNMF

Disclaimer

This presentation (**Presentation**) contains summary information about Amplia Therapeutics Limited ACN 165 160 841 and its subsidiaries (the **Company** or **Amplia**) which is current as at 30 Jun 2026. By attending an investor presentation or briefing, or accepting, accessing or reviewing this Presentation, you acknowledge and agree to the terms set out below.

Summary Information: This Presentation has been prepared for information purposes only and is a summary only. It should be read in conjunction with Amplia's most recent financial report and other periodic and continuous disclosure information lodged with the Australian Securities Exchange (ASX), which is available at www.asx.com.au. Subject only to any legal obligation to do so, the Company does not have any obligation to correct or update the content of this Presentation.

Not financial product advice: This Presentation does not, and does not purport to, contain all information necessary to make an investment decision, is not intended as investment or financial advice (nor tax, accounting or legal advice) and must not be relied upon as such. This Presentation does not take into account the investment objectives, financial situation or needs of any particular investor. Investors are encouraged to seek independent professional advice when deciding if an investment in the Company is appropriate. The Company is not licensed to provide financial product advice in respect of its own securities. This Presentation is not a prospectus, product disclosure statement or other offering document under Australian law (or any other law). It is not, and does not constitute, an invitation or offer of securities for subscription, purchase or sale in any jurisdiction.

Investment risk and past performance: An investment in Amplia shares is subject to known and unknown risks, some of which are beyond the control of the Company and its directors. The Company does not guarantee any particular rate of return or the performance of Amplia. Past performance is not, and should not be relied on as being, indicative of future performance.

Future performance and forward-looking statements: This Presentation includes forward looking statements, which can generally be identified by the use of words such as "may", "will", "expect", "intend", "plan", "estimate", "anticipate", "outlook", "forecast" and "guidance", or other similar words. They may include, without limitation, statements regarding plans, strategies and objectives and anticipated business developments. Forward-looking statements inherently involve known and unknown risks, uncertainties and other factors that may cause Amplia's actual results, performance and achievements to differ materially from statements in this Presentation.

Forward-looking statements are based on the Company's good faith assumptions as to the financial market, regulatory and other relevant environments that will exist and affect Amplia's business and operations in the future. The Company does not give any assurance that the assumptions will prove to be correct. There may be other factors that could cause actual results or events not to be as anticipated, and many events are beyond the reasonable control of the Company. Readers are cautioned not to place undue reliance on forward-looking statements. Forward-looking statements in this Presentation are only made as at the date of this Presentation and the Company does not undertake any obligation to publicly update or revise any of the forward-looking statements or to advise of any change in assumptions on which any such statement is based.

Industry data and third party information: Industry data and third party information used in this Presentation may have been obtained from research, surveys, reports or studies conducted by third parties, including industry or general publications. Neither Amplia not its representatives have independently verified any such market or industry data.

Financial Information: This Presentation contains historical financial information based on the Company's results for the 12 month period ending 30 Jun 2026 and management accounts to 30 Jun 2026. All financial information disclosed in this Presentation is presented in Australian dollars unless otherwise noted. Any discrepancies between totals and sums of components in tables and figures contained in this Presentation are due to rounding.

Disclaimer: To the maximum extent permitted by law, Amplia and its officers, directors, employees, agents and advisers: (1) disclaim all responsibility and liability (including, without limitation, any liability arising from fault, negligence or negligent misstatement) for any loss arising from this Presentation or reliance on anything contained in or omitted from it or otherwise arising in connection with this Presentation; (2) disclaim any obligation or undertaking to release any update or revision to the information in this Presentation to reflect any change in expectations or assumptions; and (3) do not make any representation or warranty, express or implied, as to the accuracy, reliability, completeness of the information in this Presentation or that this Presentation contains all material information about Amplia or that a prospective investor or purchaser may require in evaluating a possible investment in Amplia or acquisition of shares, or the likelihood of fulfilment of any forward-looking statement.

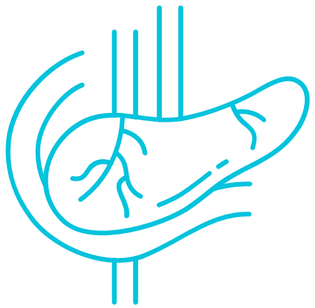
Amplia THERAPEUTICS

The FAK Opportunity

A large and unmet clinical need exists in cancers where FAK is overexpressed

Lead program

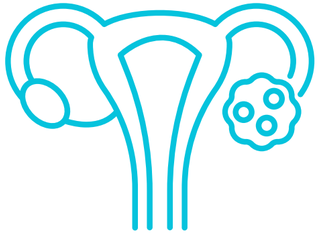
Pancreatic Cancer



US$3.2b 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$5.8b market[2]

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

kRAS Combinations

US$7.8b market (by 2035)[3]

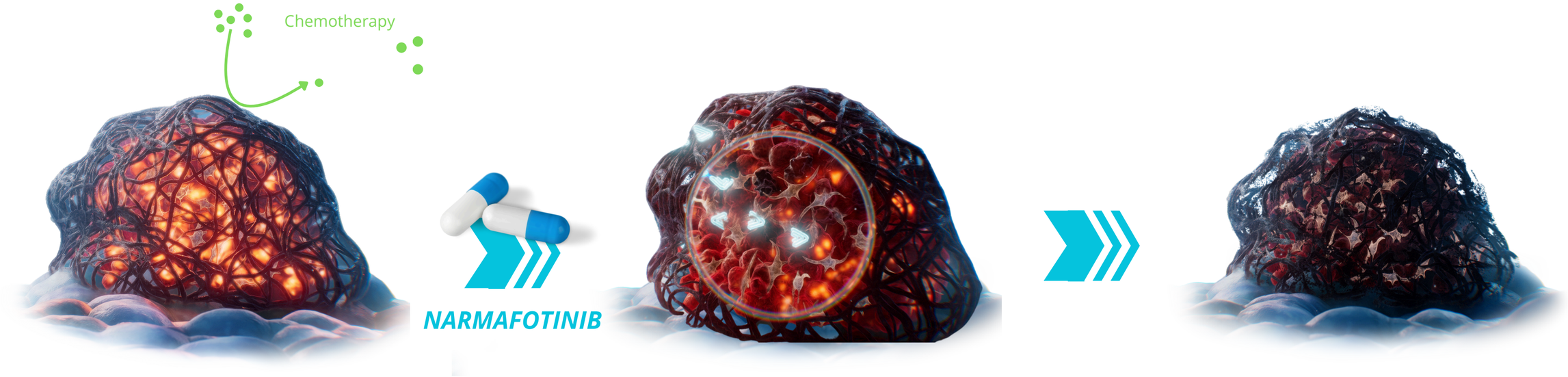
- Emerging opportunity
  60+ kRAS drugs in development
- Preclinical ATX models show narmafotinib can improve kRAS inhibitors

1. 2025 8MM's, Clarivate/ Nature Reviews Drug Discovery 25, 416-417 (2026); 2. Estimated for 2025, Business Research Company 2024; 3. Estimated for 7MM, Delveinsight 2025

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

Amplia THERAPEUTICS

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

Amplia THERAPEUTICS

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

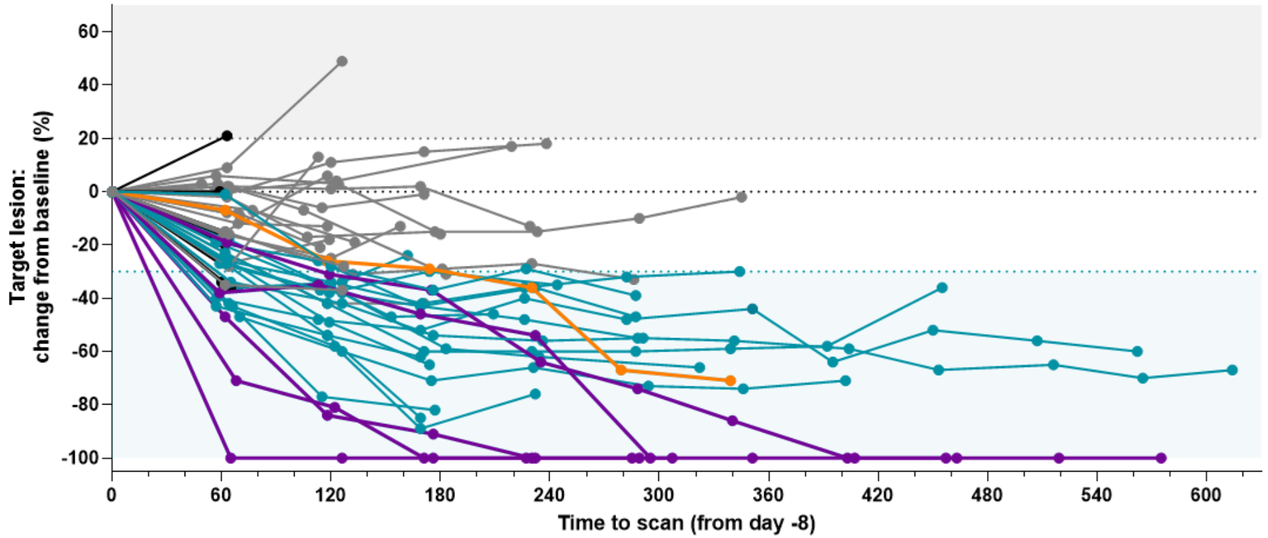
Main narma TEAEs: nausea, vomiting, diarrhoea

Amplia THERAPEUTICS

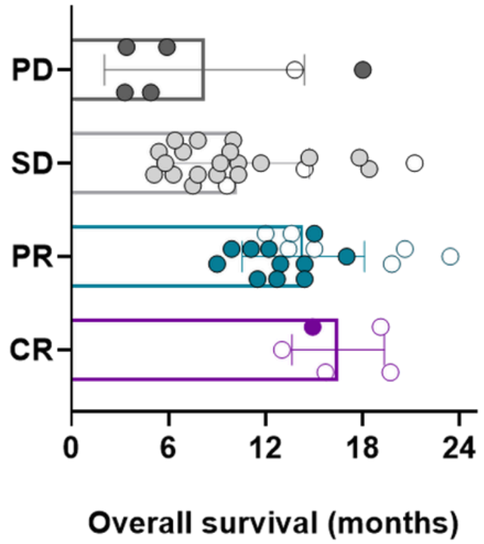
ACCENT Pancreatic Cancer Trial

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

Amplia THERAPEUTICS

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

Details to be confimed

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

Details to be confimed

ACCENT Reg - P3

Pivotal registrational study conducted with the dose selected from P2b

Designed to support FDA submission for narmafotinib approval in pancreatic cancer

Amplia THERAPEUTICS

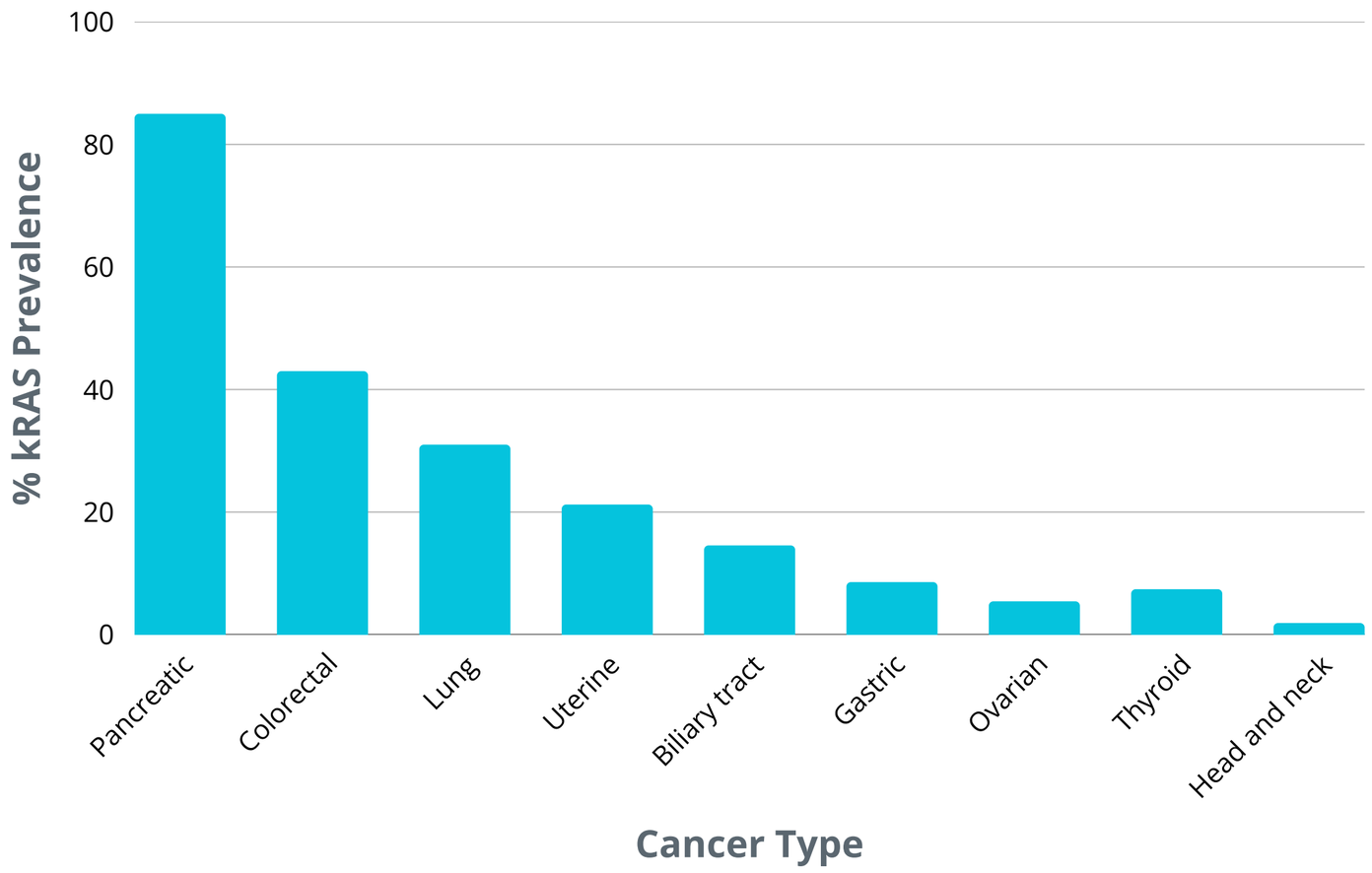
kRAS Inhibitors

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

Amplia THERAPEUTICS

kRAS mutations prevalent in cancer

Significant cancer driver in multiple indications



Clinical studies demonstrating good activity but **resistance and tolerability a significant issue**

A market growing at 35% per year
From $526M today to a projected $7.8B by 2034 across major markets[1]

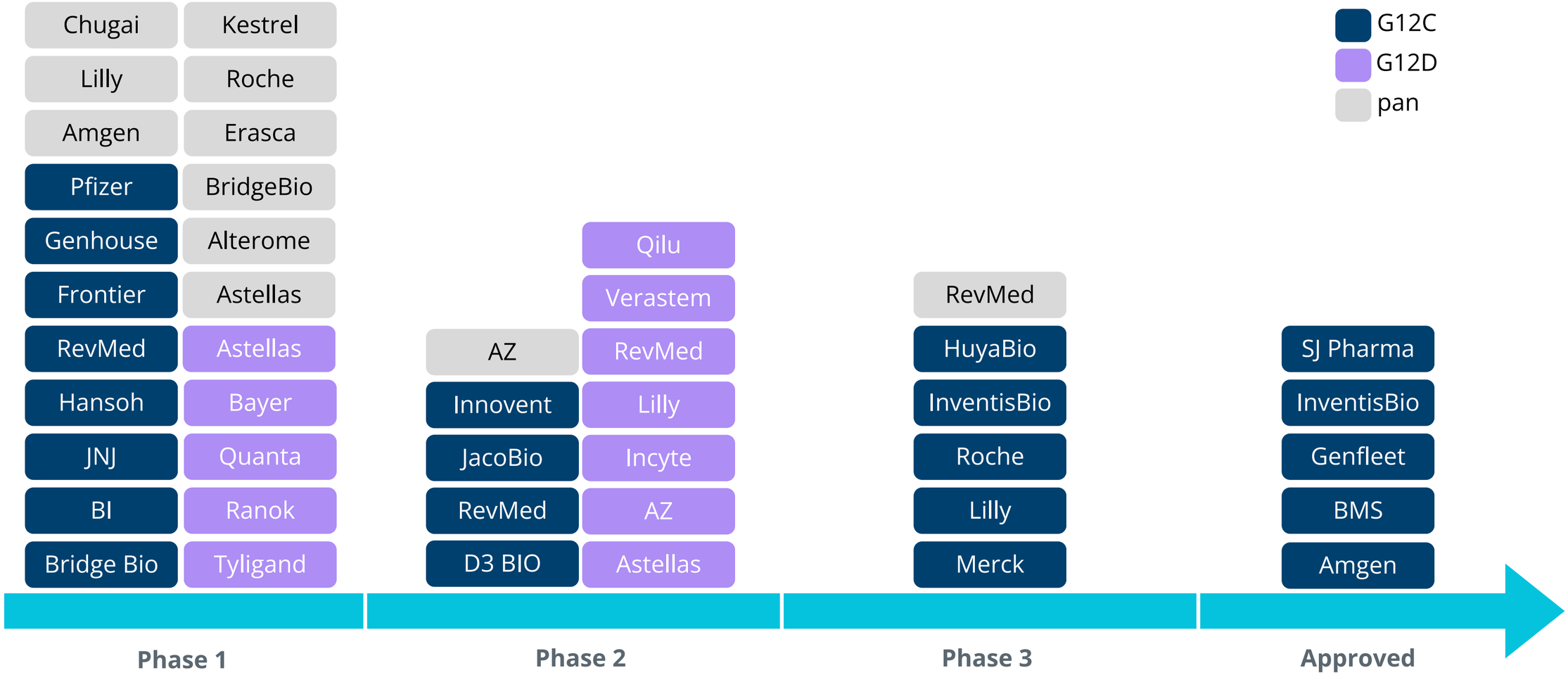
60+ kRAS drugs now in clinical development
Across pancreatic cancer, NSCLC, colorectal cancer and others

1. https://www.delveinsight.com/report-store/kras-inhibitors-market

Amplia THERAPEUTICS

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.

[1]Charité – Universitätsmedizin Berlin, Institute of Pathology, Berlin, Germany
[2]German Cancer Consortium (DKTK), Partner Site Berlin, German Cancer Research Center (DKFZ), Heidelberg, Germany
[3]Lineberger Comprehensive Cancer Center, University of North Carolina at Chapel Hill, Chapel Hill, NC 27599, USA
[4]Department of Pharmacology, University of North Carolina at Chapel Hill, Chapel Hill, NC 27599, USA

*Correspondence:
Bjoern.Papke@charite.de (B. Papke).

Trends in Cancer, February 2025, Vol. 11, No. 2 https://doi.org/10.1016/j.trecan.2024.11.009 91

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

Amplia THERAPEUTICS

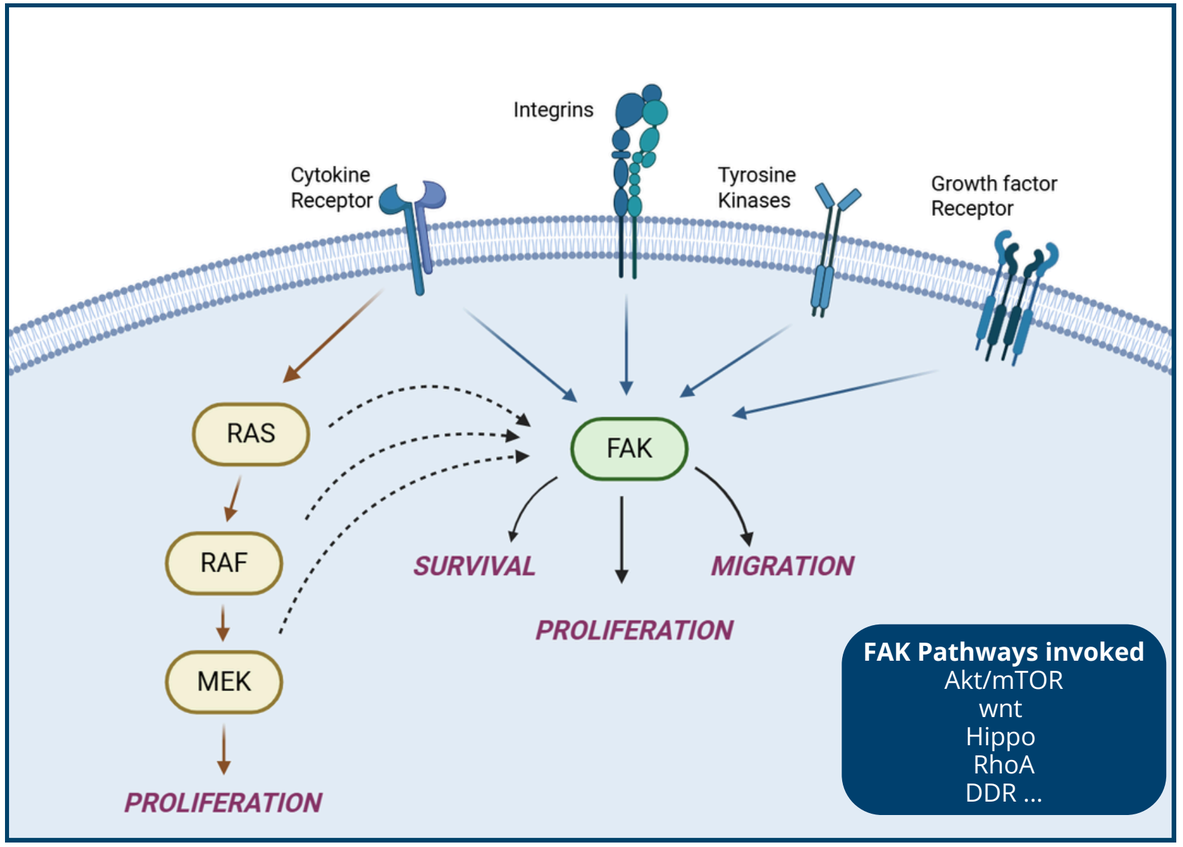
Narmafotinib and kRAS inhibition

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



Amplia THERAPEUTICS

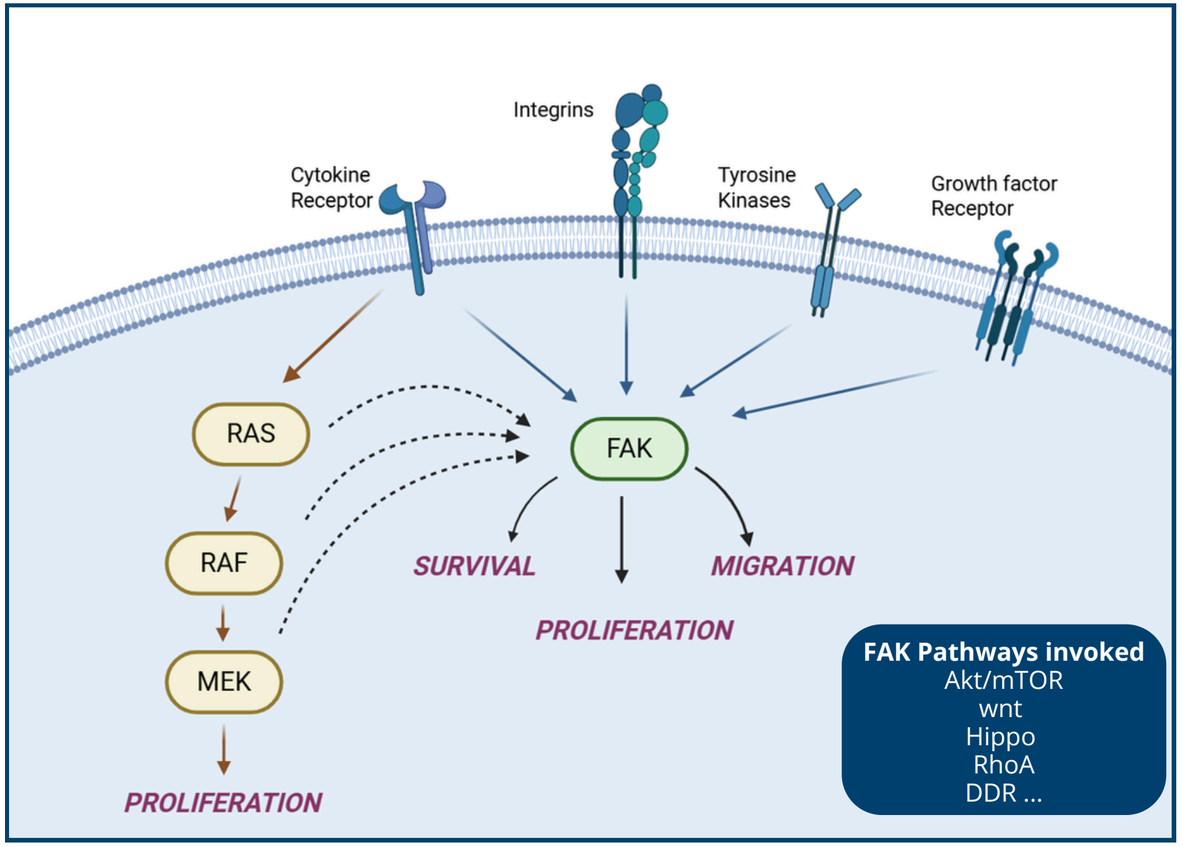
Narmafotinib and kRAS inhibition

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



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%

Amplia THERAPEUTICS

Lilly Collaboration - Detail

Enhancing efficacy for next-generation kRAS G12C inhibitor

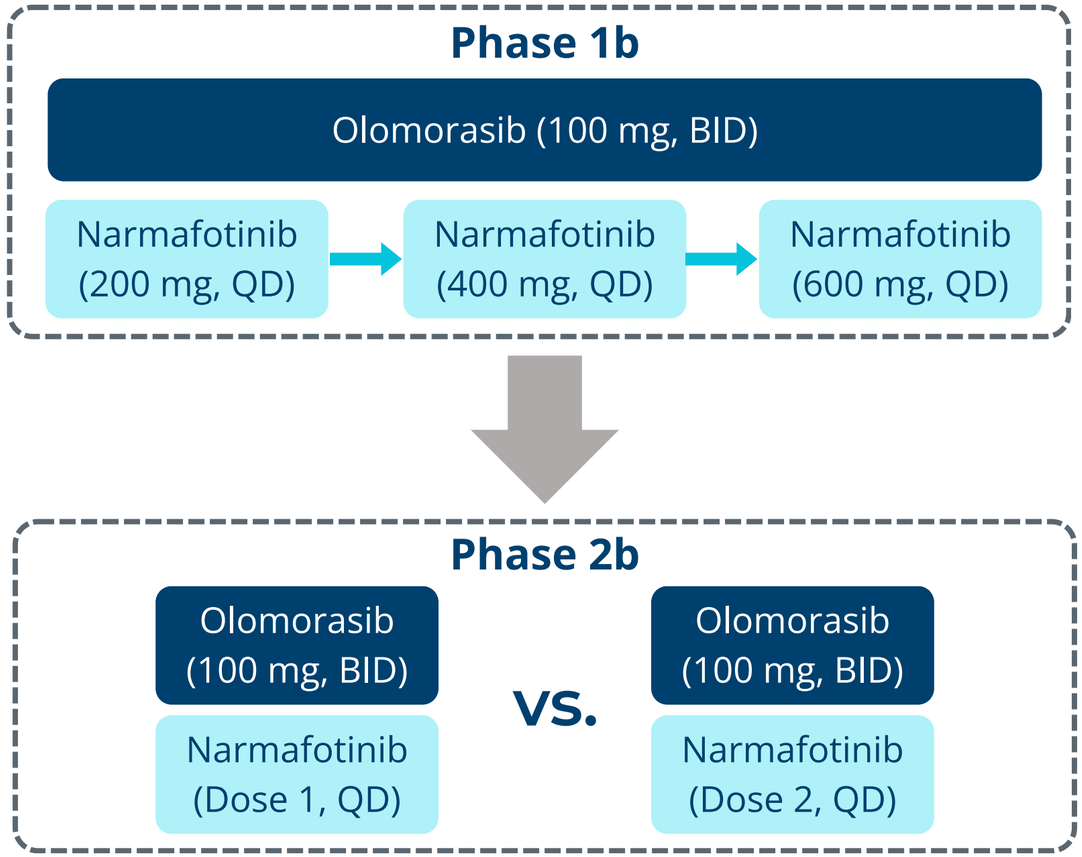
Previously treated KRAS G12C-mutant advanced or metastatic NSCLC

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



Amplia THERAPEUTICS

Lilly Collaboration

Important validation of the potential of our science

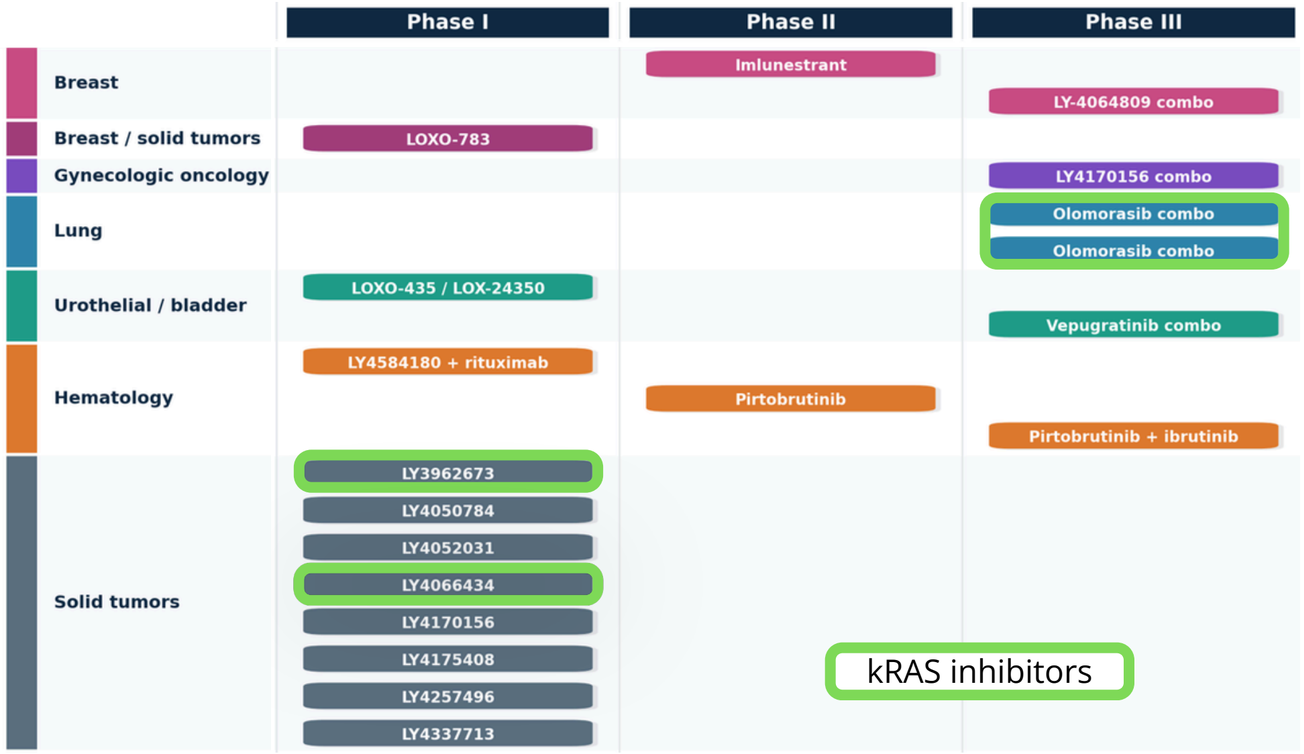
Eli Lilly and Co.

- 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

Amplia THERAPEUTICS

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

Amplia THERAPEUTICS

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

→

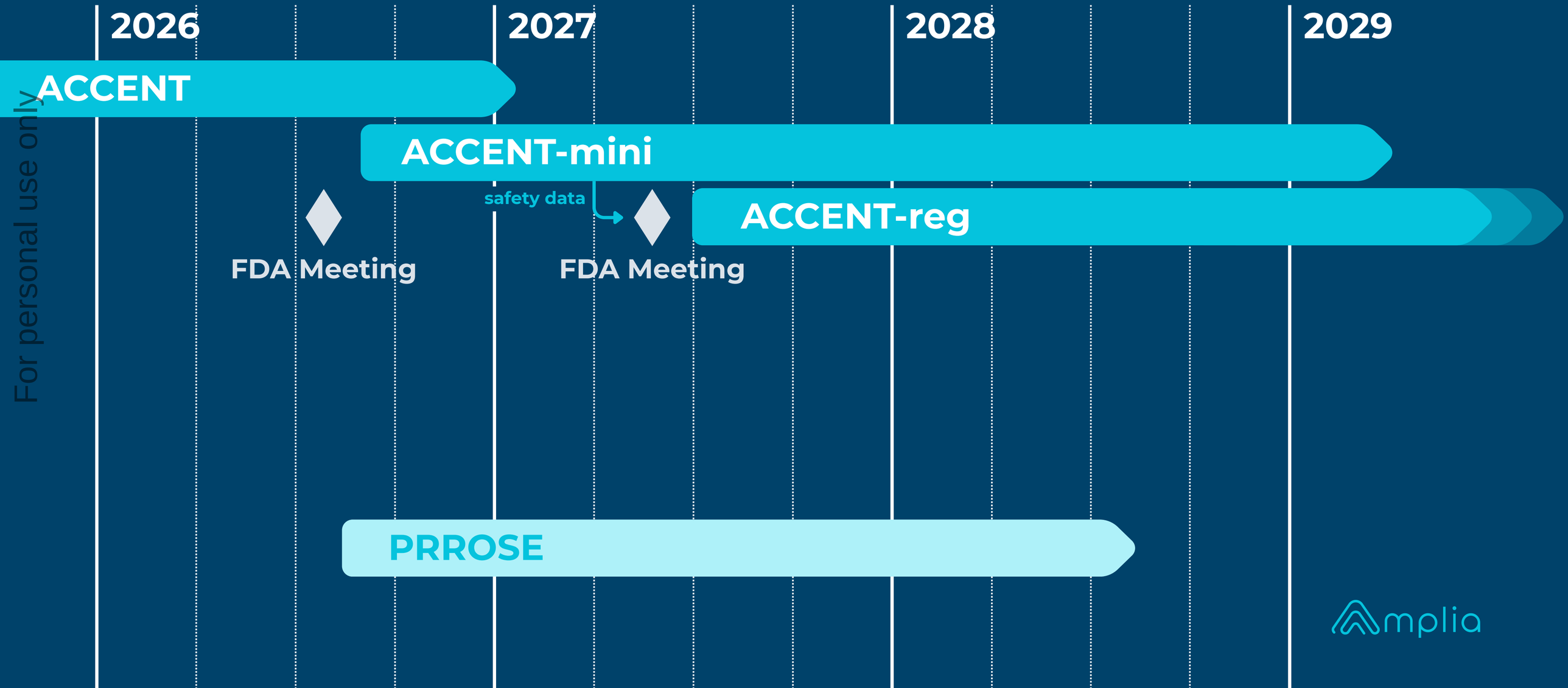
Endpoints
Safety, surgical eligibility
tissue + biomarkers

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

ANZ GOG

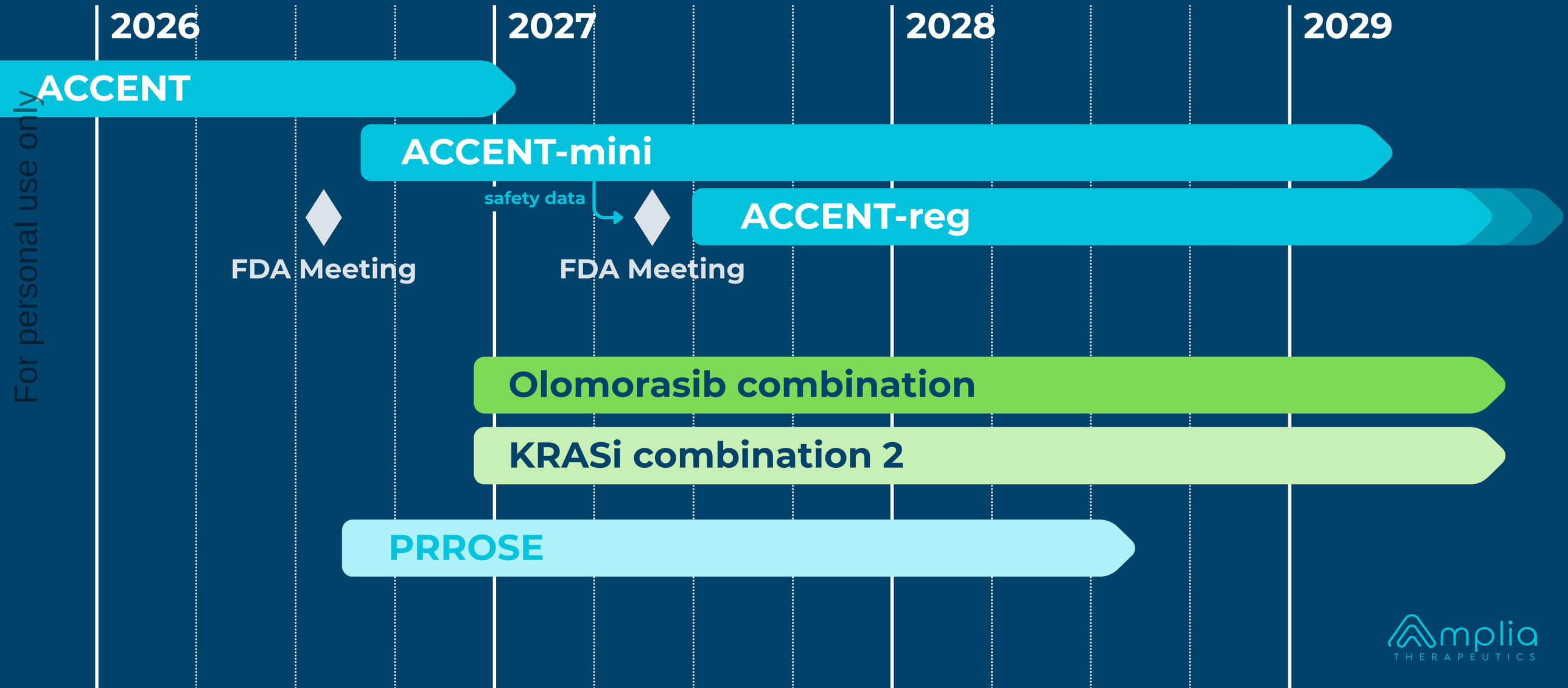
Amplia THERAPEUTICS

Development Timeline



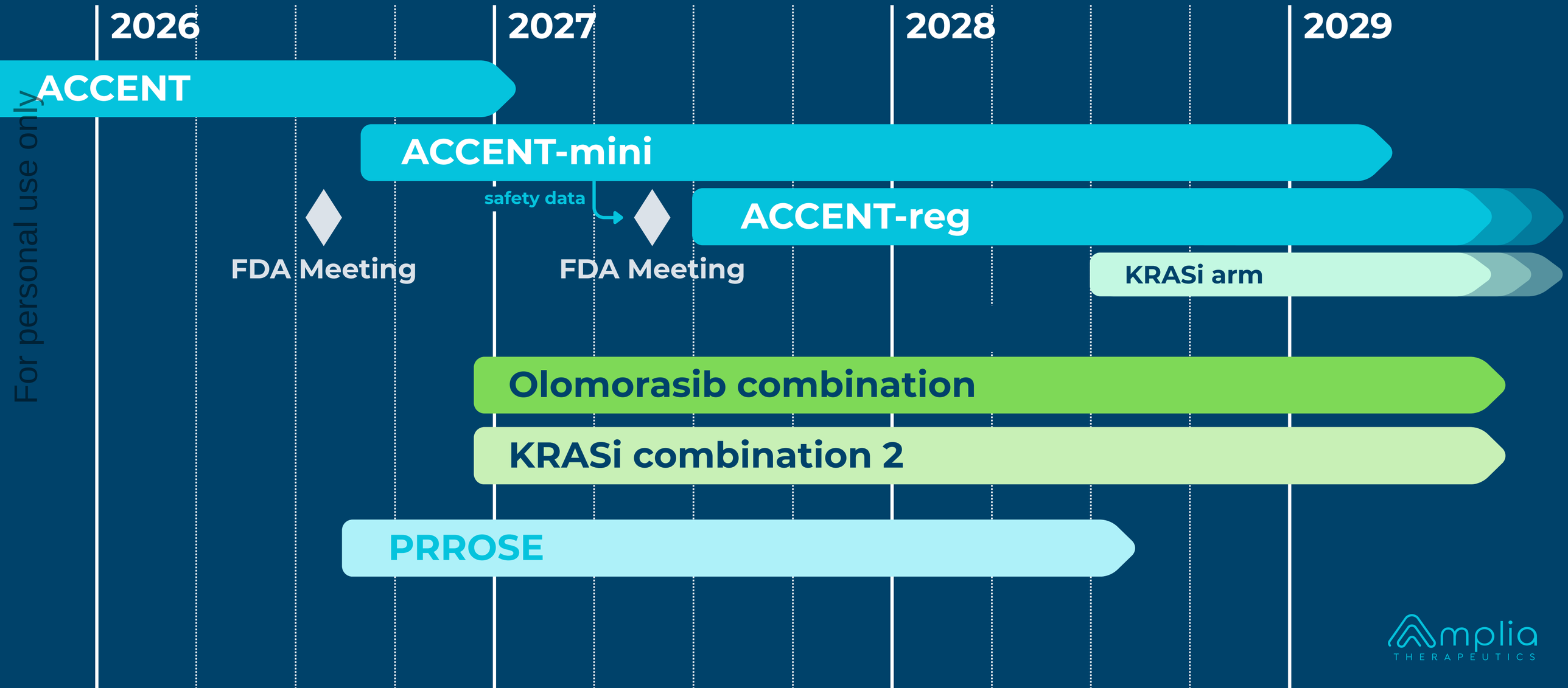
Amplia

Development Timeline



Amplia THERAPEUTICS

Development Timeline



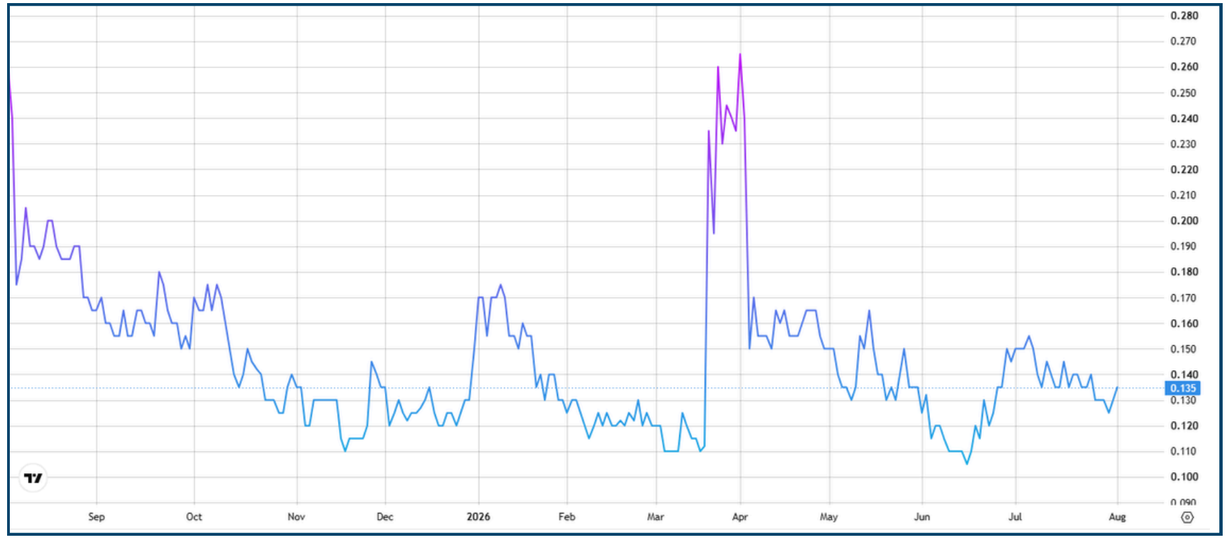
Amplia THERAPEUTICS

Corporate Summary

ASX:ATX | OTCQB:INNMF

Share price (04-Aug-2026)	A$0.155
Shares on issue	513.3M
Market cap (04-Aug-2026)	A$79.56M
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

Jane Bell AM

LLB LLM (Lond) FAICD

Chair

Monash Health

JESSIE MCPHERSON PRIVATE HOSPITAL

mesoblast

Warwick Tong

MB ChB MPP GAICD

Director

gsk

Surface Logix

Cancer Therapeutics CRC

Robert Peach

PhD

Director

biogen idec

receptos

Bristol Myers Squibb®

Brett Carter

BSc MBA GAICD

Director

gsk

Cancer Therapeutics CRC

Medicines Development for Global Health

Chris Burns

PhD GAICD FAHMS

CEO and MD

Pfizer

cytopia

DEVELOPING BETTER DRUGS

YM BIOSCIENCES INC.

SENIOR MANAGEMENT

Rhiannon Jones

PhD GAICD

COO

Cancer Therapeutics CRC

Jason Lickliter

MBBS FRACP

CMO

Nucleus Network

Hamish George

BCom MA CA GIA(Cert)

CFO

Bio101.

Jonathan Barlow

LLB BSc GDipBA GAICD

Head of BD

Telix

Amplia THERAPEUTICS

Thank You

Chris Burns PhD GAICD FAHMS
CEO and MD

chris@ampliatx.com

Rhiannon Jones PhD GAICD
COO

rhiannon@ampliatx.com

Amplia Therapeutics Limited
ASX: ATX | OTCQB: INNMF
info@ampliatx.com

ampliatx.com