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ASX RELEASE

AMPLIA PRESENTS AT CG DRUG AND DEVICE CONFERENCE 2026

Melbourne, Australia: Amplia Therapeutics Limited (ASX:ATX; OTCQB:INNMF), is pleased to release CEO and Managing Director Dr Chris Burns' presentation to the Canaccord Genuity Drug and Device conference in Noosa, QLD.

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

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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.



NARMAFOTINIB – PANCREATIC CANCER AND BEYOND

October 2026

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ASX: ATX | OTCQB: INNMF



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Amplia Therapeutics

Improving treatment options for hard-to-treat cancers



Listed biotech company (ASX:ATX; OTCQB:INNMF) developing **narmafotinib: a FAK inhibitor that decreases fibrosis and reverses adaptive resistance** limiting new and existing cancer therapies; currently in Phase 2 for first-line treatment of advanced pancreatic cancer.



Combination benefit proven in clinical studies: complete responses in metastatic pancreatic cancer (ACCENT Trial) and favorable tolerability profile, when combined with chemotherapy.



Potentially synergistic with kRAS inhibitors: collaboration with Lilly to explore combination of narmafotinib with kRAS G12C inhibitor for the treatment of metastatic non-small cell lung cancer.



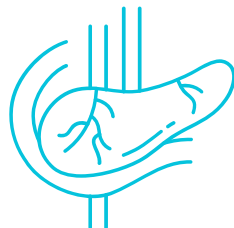
Parallel development paths underway: registrational trial in pancreatic cancer to start imminently; **clinical trial collaborations** used as an entry point for high value indications and partnerships.

The FAK Opportunity

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

Lead program

Pancreatic Cancer



US\$2.5b market¹

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 market²

PRROSE Trial
HGSOC patients | ANZGOG

High FAK expression
1 in 5 don't respond to chemo

kRAS Combinations



US\$32b lung cancer market³
US\$19b CRC market⁴

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

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

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

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 Oct 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>

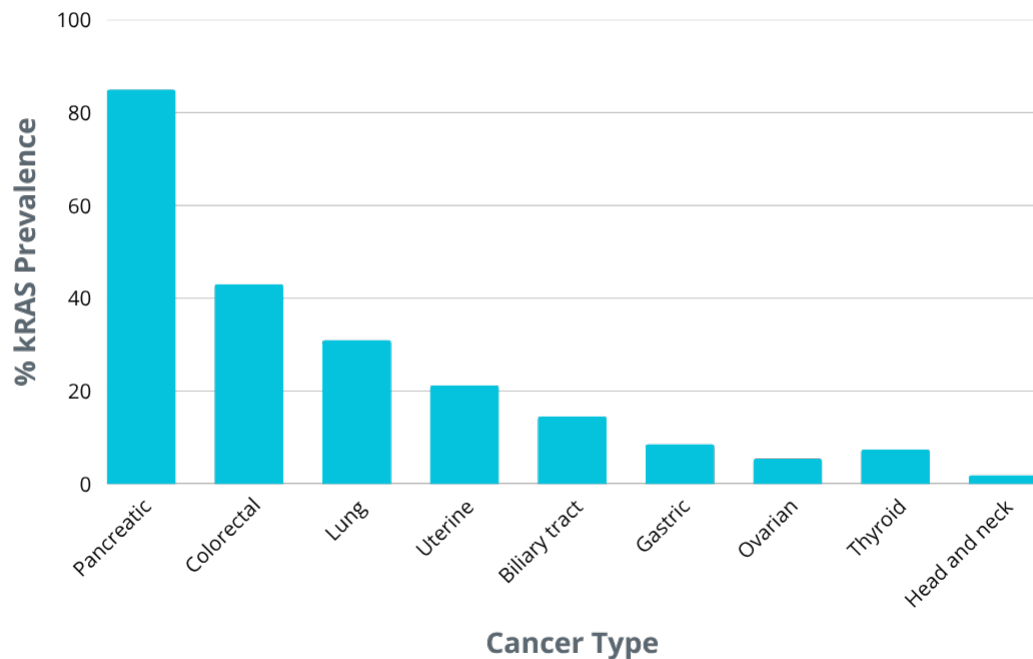


Combinations with kRAS inhibitors

FAK inhibition offers significant benefit

kRAS mutations prevalent in cancer

Significant cancer driver in multiple indications



Clinical studies demonstrating good activity



80+ kRAS drugs now in clinical development

- Biotech and Pharma
- Across pancreatic cancer, NSCLC, colorectal cancer and others



Resistance and tolerability significantly limits long term treatment

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 Isenmann^{1,2}, Christine Sers^{1,2}, Channing J. Der^{1,3,4}, and Björn 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 KRASG12C 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, Shokat's group identified the first mutant-specific inhibitor targeting KRAS^{G12C}. The breakthrough highest precision oncology 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^{G12V}), as well as pan-KRAS inhibitors or RAS/RAF/MEK/ERK inhibitors, are entering clinical trials and demonstrating promising results.

With the growing number of KRAS inhibitors, patients with mutant KRAS are looking 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 (45%, 85%, and 31%, respectively) (Figure 1A) [7]. By contrast, HRAS mutations are commonly linked to skin cancer (including melanoma) (6%), bladder cancer (6%), and head and neck cancer (7%), while KRAS mutations are prominent in melanoma (22%), 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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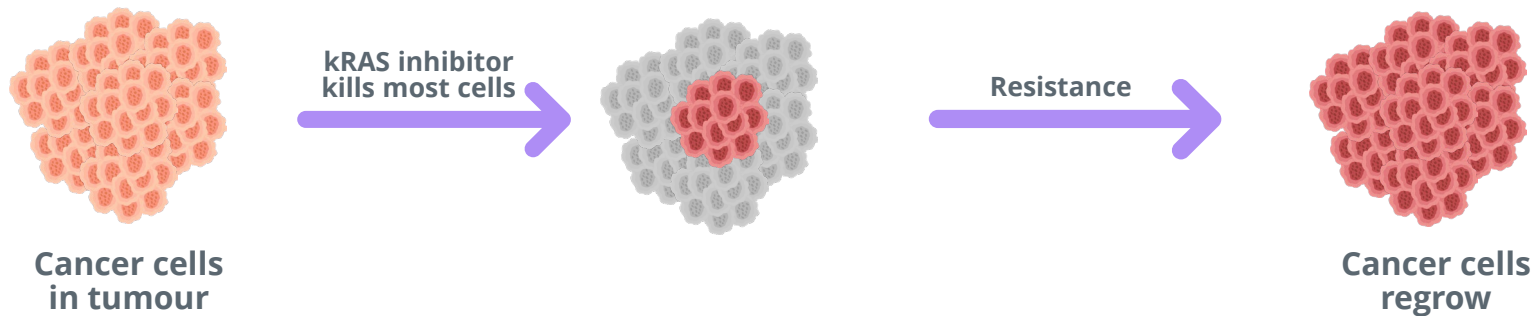


Trends in Cancer, February 2025, Vol. 11, No. 2 <https://doi.org/10.1016/j.trecan.2025.100002> 91
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Trends in Cancer, February 2025, Vol. 11, No. 2

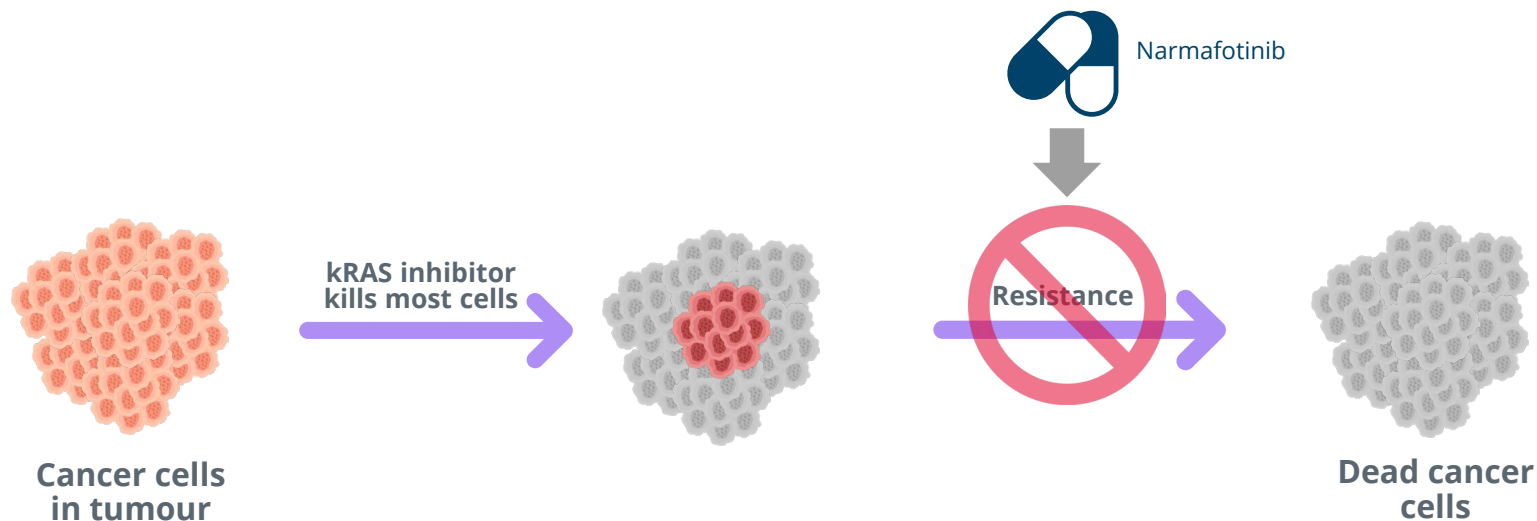
Narmafotinib target processes leading to resistance

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



Narmafotinib target processes leading to resistance

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



Narmafotinib's mechanism appears **agnostic to tumour type or kRAS mutation** and has been demonstrated with a **variety of kRAS inhibitors**

Lilly Collaboration

Enhancing efficacy of next-generation G12C inhibitor

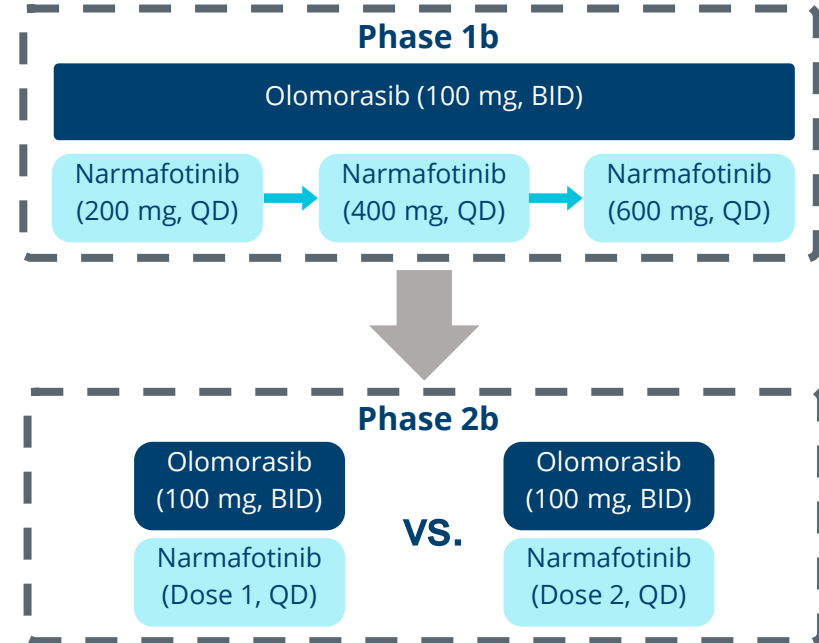
Previously treated KRAS G12C-mutant advanced or metastatic NSCLC

Phase 1b/2b study

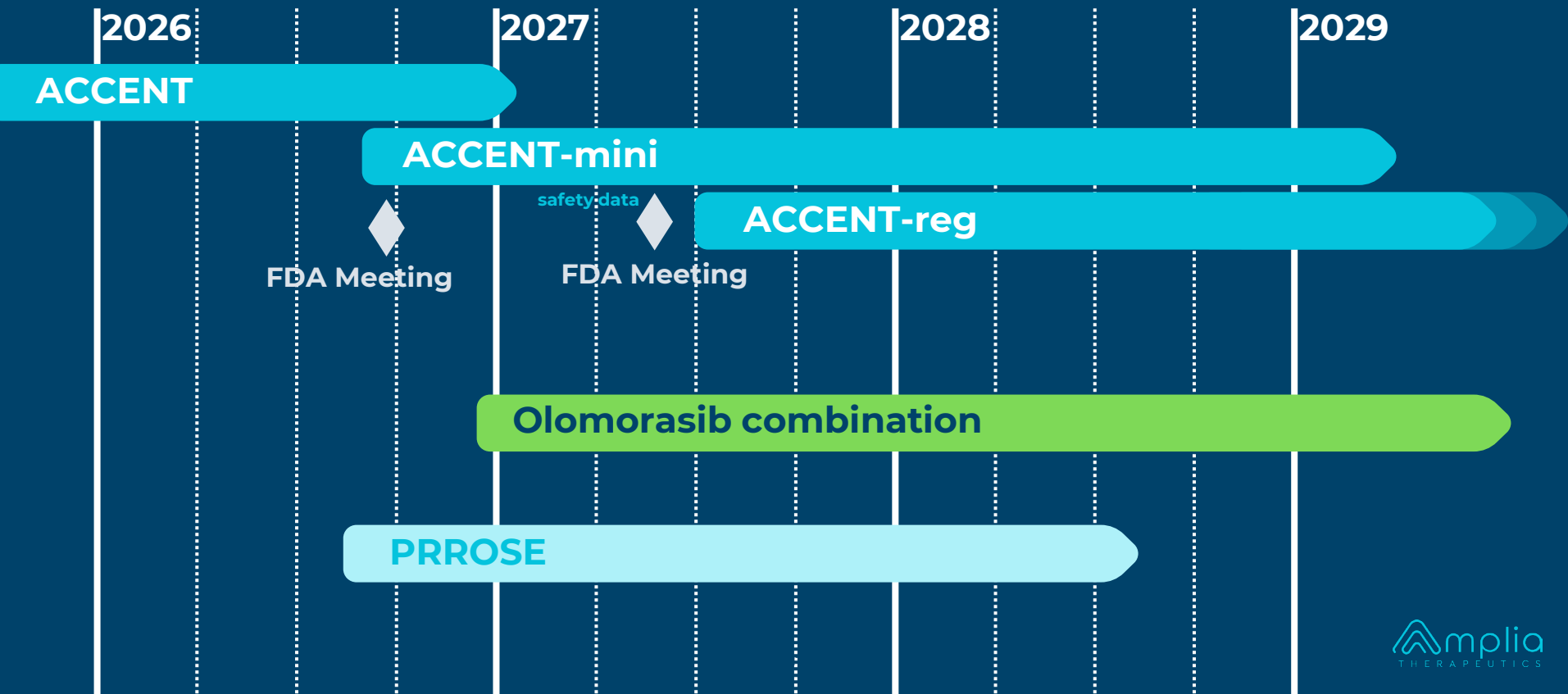
- **Dose escalation of narmafotinib** with standard dose olomorasib (approx. 20 pts)
- **Randomised 2-dose comparison** – following Project Optimus guidelines (approx. 40 pts.)
- Australian and US sites

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



Estimated Development Timeline



Forthcoming Catalysts

Timing	Milestone
Q4 2026	FDA Meeting discussing development
Q4 2026	First patient dosed ACCENT-mini
Q4 2026	First patient dosed PRROSE
Q4 2026	Ethics submission Lilly trial
Q1 2027	ACCENT-mini key data available
Q1 2027	First patient dosed Lilly trial
Q2 2027	FDA meeting finalising registration trial
H2 2027	Preliminary data - PRROSE and Lilly trials

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