EVOLUTION CAPITAL

ATX | UPDATE REPORT | HEALTHCARE

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AMPLICITY Halted; Focus Sharpens on Gem/Abraxane & KRAS Optionality

Amplia Therapeutics Ltd

Amplia has announced it is permanently halting recruitment in the AMPLICITY clinical trial following three protocol-defined dose-limiting toxicities (DLTs), none of which were attributed to narmafotinib. All three events relate to the modified FOLFIRINOX (mFOLFIRINOX) chemotherapy backbone, consistent with the regimen's well-documented toxicity profile. Eight patients had been dosed; five remain on study and will continue receiving the narmafotinib/mFOLFIRINOX combination under ongoing safety monitoring. Management will redirect resources toward combinations other than FOLFIRINOX, explicitly flagging an intent to pursue studies with new targeted agents in pancreatic cancer.

ACCENT Final Data Recently Announced

Last month's mature ACCENT readout established the core data package on which the investment case now stands: an independently centrally-adjudicated 7.8% confirmed CR rate (5/64), an ORR of 35.9%, and a median overall survival of 11.1 months, matching the FDA-approved NALIRIFOX regimen with a simpler two-drug backbone and a substantially cleaner tolerability profile. That dataset is undisturbed by today's announcement and remains the foundation for partnering discussions and the next-stage clinical strategy.

Outlook

Yesterday's halt is disappointing but not thesis breaking. AMPLICITY was the secondary pillar, designed to broaden suitability to fitter, higher-performance-status patients who typically receive FOLFIRINOX. The safety signal here is clean from narmafotinib's perspective: the chemotherapy backbone failed the tolerability test, not the drug. We maintain our Speculative Buy recommendation.

We await the March quarter Appendix 4C, expected later this month, for an updated cash figure; with AMPLICITY no longer consuming trial spend, the existing runway should extend modestly. A pivotal Phase 2b/3 study of narmafotinib plus gem/Abraxane will likely require more capital than Amplia currently holds, and the company will likely need to raise further funds, whether via partnership, equity, or a combination, to fund a registrational study to completion.

Catalyst	Est. Timing
AACR annual meeting presentation	17-22 April 2026
FDA interactions on Phase 2b/3 design and potential accelerated approval pathway	H1 2026
March quarter Appendix 4C (cash position update)	Late April 2026
Potential phase 2b/3 trial design announcement	H2 2026
Partnership / licensing announcement	TBD

Recommendation	**Spec Buy**
Previous Close	**$0.15**
Fair Valuation	**UR; Prev. $0.47**
TSR	**UR**

Company Profile (on Prev Close)

Market Cap	$77.0M
Enterprise Value	$45.5M
SOI (undiluted)	513.1M
Free Float	89.2%
ADV (3-month)	$760.1k
52-Week Range	$0.049 - $0.425

Price Performance

Company Overview

Amplia Therapeutics (ASX: ATX) is a clinical-stage biotechnology company developing targeted therapies for aggressive, treatment-resistant cancers. Its lead asset, narmafotinib (AMP945), is a highly potent and selective FAK inhibitor designed to enhance the efficacy of chemotherapy by dismantling the tumour's fibrotic and immunosuppressive defences. With FDA Fast Track and Orphan Drug designations secured, and an AACR presentation forthcoming, Amplia is positioned at a key value inflection point ahead of anticipated partnership discussions.

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Coverage:

Initiation	*9 Jul 2025*
Update	*7 Aug 2025*
Update	*10 Oct 2025*
Update	*23 Mar 2026*

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- *ATX*

AMPLICITY Update

Eight patients were enrolled across US and Australian sites in the Phase 1b/2 AMPLICITY study, which combined daily narmafotinib dosing with mFOLFIRINOX at routine cycle doses. Three DLTs have been observed. All three were determined to be related to the chemotherapy backbone rather than narmafotinib. Five of the eight enrolled patients remain on treatment. Four of the eight achieved stable disease at their first (2-month) scan, with one patient subsequently converting to a partial response at the 4-month scan. Company commentary flags an expected shift in clinical practice toward less toxic chemotherapy regimens. Due to this and the DLT signal, the Company has decided to discontinue recruitment rather than attempt dose modifications.

The strategic read is straightforward. FOLFIRINOX is a four-drug cytotoxic regimen with a well-characterised toxicity ceiling: its pivotal studies reported Grade 3+ neutropenia rates around 46% and meaningful rates of diarrhoea and neuropathy. Layering any additional agent, even one as clean as narmafotinib, onto a regimen already at its tolerability limits carries inherent risk. The fact that none of the DLTs were attributable to narmafotinib reinforces the favourable safety profile established in ACCENT, where Grade 3+ peripheral neuropathy was reported at just 3.6% versus 17% for gem/Abraxane alone in MPACT.

Strategic Pivot

Accent Follow-Up Becomes the Sole Near-Term Priority

With AMPLICITY shelved, Amplia's clinical strategy concentrates entirely on narmafotinib plus gem/Abraxane. This is where the data is strongest: ACCENT's mature readout delivered a centrally-adjudicated 7.8% confirmed CR rate (5/64), an ORR of 35.9%, and a median overall survival of 11.1 months, which is identical to NALIRIFOX with a simpler two-drug backbone and a dramatically superior tolerability profile (Grade 3+ diarrhoea 5.5% vs 20.3%; nausea 3.6% vs 11.9%).

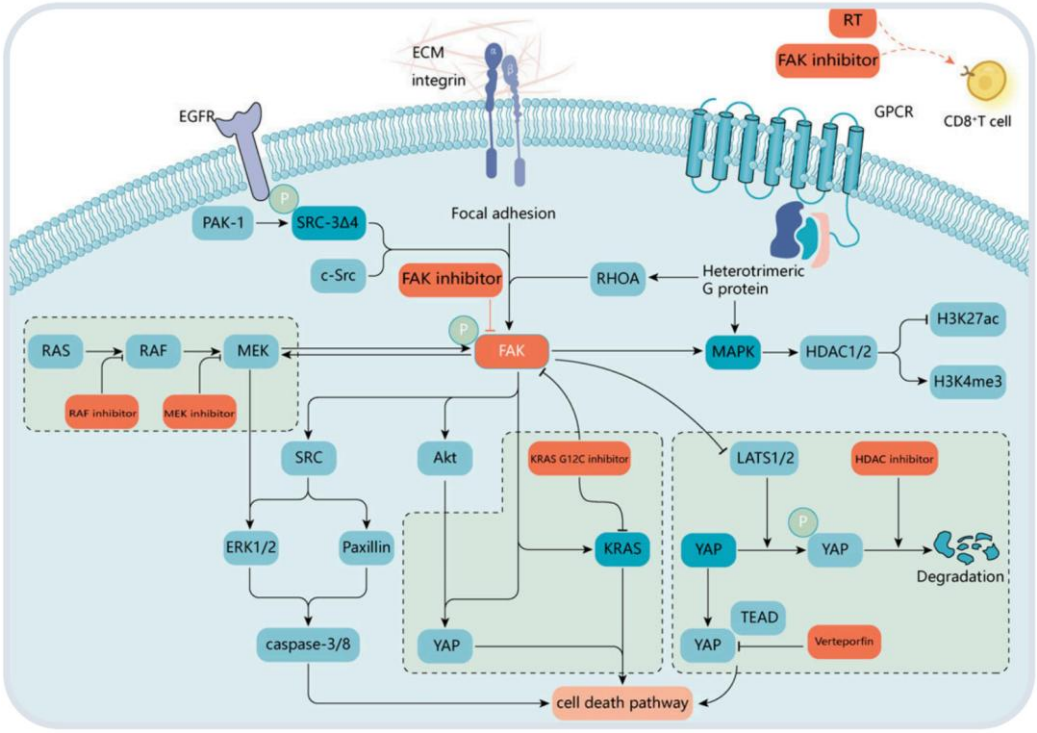
The logical next step is a pivotal Phase 2b/3 study of narmafotinib plus gem/Abraxane in first-line metastatic pancreatic cancer. The final trial design will depend on FDA feedback, which we expect will be informed by regulatory interactions post-AACR presentation later in April. At a high level, investors can expect the Company to proceed with a randomised Phase 2b/3 with OS as the primary endpoint, powered to detect a clinically meaningful improvement over gem/Abraxane alone. The unprecedented CR rate may also support consideration of an accelerated approval pathway; a question we flagged in our March note and which will become clearer following regulatory engagement.

Consolidating around a single study with an already-validated chemotherapy backbone also simplifies the partnering narrative. Pharmaceutical partners pricing a deal around narmafotinib will now focus on a single, cleaner dataset (ACCENT) rather than waiting on a parallel FOLFIRINOX readout that was always going to be secondary in the investment case.

Unlocking the KRAS Opportunity

Yesterday’s developments frees Amplia to pursue a large source of unmodelled upside in the narmafotinib story: combination with KRAS inhibitors. KRAS is mutated in approximately 90% of pancreatic cancers and sits at the top of the RAS-RAF-MEK-ERK (MAPK) signalling cascade. FAK is a parallel signalling hub that intersects with, and provides a feedback escape route from, MAPK blockade. When KRAS is inhibited upstream, whether via direct KRAS G12C/G12D agents or via MEK inhibition, tumours upregulate FAK signalling as a resistance mechanism. Dual blockade is therefore mechanistically synergistic rather than merely additive.

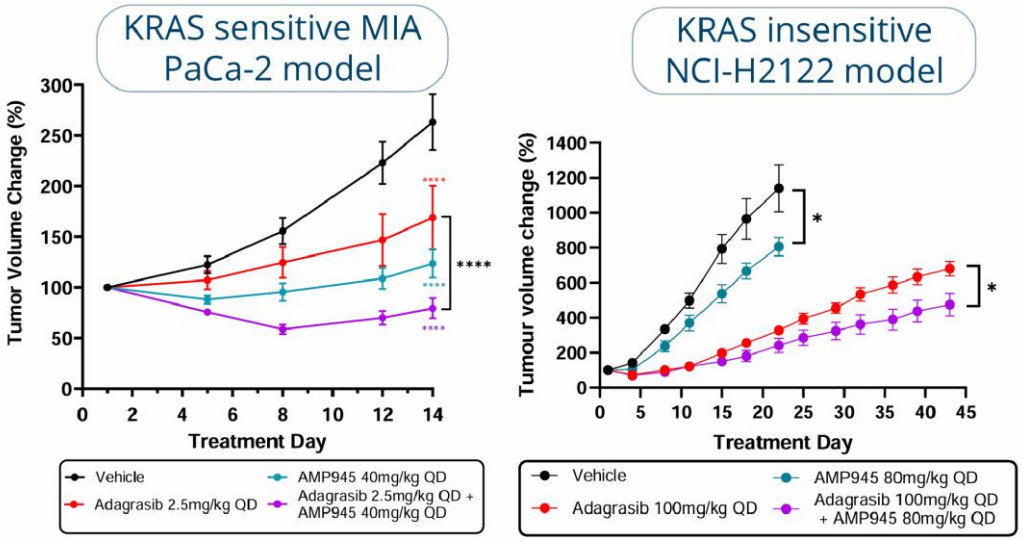
Figure 1: The FAK-KRAS signalling axis. FAK sits at the intersection of integrin-mediated adhesion signalling and the canonical RAS-RAF-MEK-ERK (MAPK) growth pathway. Source: company materials.



The diagram illustrates why FAK and KRAS are mechanistically intertwined rather than parallel targets. KRAS sits at the top of the canonical MAPK cascade (RAS → RAF → MEK → ERK), the primary oncogenic growth signal in pancreatic cancer. FAK occupies a central node downstream of integrin-mediated adhesion at the cell membrane, but its signalling network feeds directly into the same downstream effectors – SRC, AKT, ERK, and KRAS itself – that drive proliferation and survival. The clinical consequence is that blocking KRAS upstream (whether with a direct KRAS G12C/G12D inhibitor or with a MEK inhibitor further down the MAPK chain) does not fully shut down the growth signal. Tumours respond to MAPK blockade by upregulating FAK activity, which reactivates the same downstream pathways via a parallel route. This is the molecular basis for acquired resistance to KRAS-targeted therapy, and the rationale for combining a FAK inhibitor with a KRAS inhibitor to close the escape hatch.

Amplia has generated direct preclinical evidence for this combination. Work presented at the AACR Special Conference on Pancreatic Cancer in January 2025 tested narmafotinib with adagrasib (Mirati's KRAS G12C inhibitor) across two models. In the KRAS-sensitive MIA PaCa-2 pancreatic model, adagrasib monotherapy slowed tumour growth (tumours still expanding ~175% over 14 days), narmafotinib alone was modestly active (~125% growth), but the combination drove tumours into regression, shrinking to approximately 60% of baseline volume by day 8 and holding suppressed thereafter, a result that was highly statistically significant. In the KRAS-insensitive NCI-H2122 lung model, adagrasib alone barely restrained tumour growth (>700% expansion by day 45), whereas the combination capped growth at approximately 500%; a statistically significant restoration of sensitivity in a resistant background. The readthrough is that narmafotinib can both deepen responses in KRAS-sensitive disease and help overcome primary resistance in KRAS-insensitive disease.

Figure 2: Narmafotinib enhances and sustains tumour responsiveness to the KRAS G12C inhibitor adagrasib in preclinical models. Source: Kinkel et al., Keystone Symposium 'Tumor Microenvironment: Metastasis and the Host', Banff, January 2025 (ATX ASX Release, 20 January 2025).



KRAS-sensitive MIA PaCa-2 pancreatic model (left): adagrasib monotherapy initially controls tumour growth but the effect plateaus as resistance emerges. Narmafotinib monotherapy is modestly active. The combination, however, drives a markedly deeper and more durable response than either agent alone, with the effect statistically significant versus both monotherapies. The implication is that FAK inhibition deepens and prolongs the benefit of KRAS blockade in tumours that are intrinsically responsive to it, addressing the well-documented problem of acquired resistance to KRAS G12C inhibitors. **KRAS-insensitive NCI-H2122 lung model** (right): adagrasib alone has minimal effect, consistent with the model's resistant phenotype. Adding narmafotinib produces a statistically significant suppression of tumour growth that neither agent achieves on its own. This points to a second, equally important use case: FAK inhibition can restore sensitivity to KRAS-targeted therapy in tumours that would otherwise not respond.

KRAS Inhibition Incumbents

The commercial thesis is externally validated by two independent actors pursuing the same mechanistic hypothesis. Verastem Oncology received FDA accelerated approval in May 2025 for its combination of defactinib (a FAK inhibitor) and avutometinib (a RAF/MEK clamp) in recurrent KRAS-mutant low-grade serous ovarian cancer. This was the first ever regulatory approval for a FAK inhibitor. Verastem's RAMP 205 trial is now progressing this combination into first-line mPDAC as a triplet with gem/Abraxane, having reported an 83% ORR (10 of 12 patients) at the recommended Phase 2 dose.

InxMed, a well-funded Chinese biotech, provides the second point of validation. Its selective FAK inhibitor ifebemtinib combined with the KRAS G12C inhibitor garsorasib has generated standout data in first-line KRAS G12C non-small cell lung cancer: 90.3% ORR, 19.4-month median duration of response, and 22.3-month median progression-free survival. In a randomised refractory KRAS G12C colorectal cancer cohort, the combination nearly doubled ORR versus garsorasib monotherapy (44.4% vs 16.7%) and extended mPFS from 4.0 to 7.7 months. InxMed has initiated a randomised Phase 3 pivotal study in first-line KRAS G12C NSCLC and is actively exploring combinations with G12D and pan-RAS inhibitors.

The implication for Amplia is material: narmafotinib has a best-in-class safety profile – a critical differentiator versus defactinib, whose broader kinase footprint includes off-target activity against CMGC-family kinases that overlap with chemotherapy toxicities. As the KRAS inhibitor class expands beyond G12C into G12D and pan-RAS variants, the demand for a tolerable, selective FAK combination partner grows in lockstep. Redirecting AMPLICITY's freed capital and clinical bandwidth toward KRAS combination work, whether via partnership with a KRAS inhibitor developer or via investigator-initiated studies, is a higher-return use of resources than continuing a toxic FOLFIRINOX study.

None of this KRAS optionality is currently captured in our rNPV model. The SOTP valuation reflects only the core first-line mPDAC opportunity with gem/Abraxane. KRAS combination work represents pure, un-modelled upside, and counterintuitively, the halt of AMPLICITY accelerates the timeline to unlocking it.

Cash Position & Outlook

With AMPLICITY no longer consuming trial costs, the effective cash runway should extend modestly, providing management breathing room to scope the next study without immediate funding pressure. A Phase 2b/3 pivotal trial in first-line mPDAC is a materially larger undertaking than either of the existing studies. We estimate the total cost of a randomised, adequately powered Phase 2b/3 study at in excess of A$40M, depending on final design. This figure excludes the potential cost of parallel KRAS combination work. Amplia's current cash position is not sufficient to fund a pivotal study to completion on a standalone basis.

Consequently, the corporate pathway from here remains dependent on one of two outcomes: a strategic partnership with a major pharmaceutical company to co-fund and co-develop the pivotal study, or a further capital raise (which, if required, would most sensibly be staged alongside partnership discussions to minimise dilution). The strength of the ACCENT data package materially improves Amplia's negotiating leverage in partnering discussions. We expect licensing interest to intensify in the weeks around the AACR presentation in April, and we continue to view a partnership outcome as the most likely funding solution for Phase 2b/3.

For the avoidance of doubt: the company is not facing a near-term funding cliff. The existing cash position, combined with the R&D tax incentive and continued cost discipline, provides sufficient runway to reach the key partnering catalysts.

Today's announcement is a setback, but a manageable one. AMPLICITY was always the secondary pillar of the investment case; the primary pillar – narmafotinib plus gem/Abraxane via ACCENT – remains intact. Strategic consolidation around the more compelling dataset simplifies the partnering narrative and frees resources for the more interesting KRAS optionality. We reiterate our Speculative Buy recommendation, with fair valuation remaining Under Review.

Income Statement *A$Ms*	FY25a	FY26	FY27	FY28	FY29
Revenue	-	-	50.00	-	100.00
Other Income	4.06	5.66	4.35	3.05	3.05
Total Revenue	**4.06**	**5.66**	**54.35**	**3.05**	**103.05**
Operating expenses	-10.49	-15.75	-13.01	-10.30	-10.03
EBITDA	**-6.43**	**-10.10**	**41.34**	**-7.25**	**93.01**
D&A	-0.09	-0.00	-0.00	-0.00	-0.00
EBIT	**-6.52**	**-10.10**	**41.34**	**-7.25**	**93.01**
Net Interest	-0.06	0.21	0.48	1.37	1.17
NPBT	**-6.57**	**-9.89**	**41.82**	**-5.88**	**94.18**
Tax expense	-	-	-	-	-
Discontinued ops	-	-	-	-	-
NPAT	**-6.57**	**-9.89**	**41.82**	**-5.88**	**94.18**

Balance Sheet *A$Ms*	FY25a	FY26	FY27	FY28	FY29
Cash	10.86	25.22	70.80	65.09	160.34
Receivables	-	-	-	-	-
Inventory	-	-	-	-	-
R&D Incentive Receivable	3.77	5.66	4.35	3.05	3.05
Other	0.29	0.51	0.39	0.27	0.27
Current assets	**14.93**	**31.38**	**75.54**	**68.41**	**163.66**
Intangibles	7.94	7.94	7.94	7.94	7.94
PPE	0.00	0.00	0.00	0.00	0.00
Other	0.07	0.07	0.07	0.07	0.07
Non-current assets	8.01	8.01	8.01	8.00	8.00
Total Assets	**22.94**	**39.39**	**83.54**	**76.42**	**171.66**
Accrued Liabilities	1.80	2.72	2.25	1.78	1.73
Borrowings	-	-	-	-	-
Lease Liabilities	0.01	-	-	-	-
Other	0.07	0.36	0.06	-	-
Current liabilities	**1.89**	**3.08**	**2.31**	**1.78**	**1.73**
Borrowings	-	-	-	-	-
Other liability	0.02	0.03	0.03	0.04	0.04
Non current liabilities	**0.02**	**0.03**	**0.03**	**0.04**	**0.04**
Total Liabilities	**1.91**	**3.10**	**2.34**	**1.81**	**1.77**
Net Assets	**21.02**	**36.29**	**81.21**	**74.60**	**169.89**
Contributed Equity	167.39	188.57	194.08	194.89	194.89
Retained earnings	-145.54	-155.43	-113.60	-119.49	-25.31
Reserves/Other	-0.83	3.14	0.73	-0.80	0.28
Total Equity	**21.02**	**36.28**	**81.21**	**74.61**	**169.87**

Statement of Cashflows *A$Ms*	FY25a	FY26e	FY27e	FY28e	FY29e
Net profit for period	-6.57	-9.89	41.82	-5.88	94.18
D&A	0.09	0.00	0.00	0.00	0.00
ΔNWC	-0.82	3.28	-2.19	-1.95	-0.05
Other	-	-	-	-	-
Operating cash flow	**-7.30**	**-6.60**	**39.63**	**-7.83**	**94.13**
Payments for PPE	-0.00	-	-	-	-
Other payments	-	-	-	-	-
Asset sale	-	-	-	-	-
Investing cash flow	**-0.00**	**-**	**-**	**-**	**-**
Equity raised	17.28	20.00	-	-	-
Transaction costs	-1.33	-1.20	-	-	-
Exercise of options	-	2.38	5.51	0.81	-
Net borrowings	-1.47	-	-	-	-
Finance costs	-0.08	-0.05	-0.05	-0.05	-0.05
Other	-0.08	0.21	0.48	1.37	1.17
Financing cash flow	**14.40**	**21.34**	**5.95**	**2.13**	**1.12**
Free cash flow	**-7.31**	**-6.60**	**39.63**	**-7.83**	**94.13**
Net cash flow	**7.10**	**14.74**	**45.58**	**-5.71**	**95.25**
Effects of exchange rate	-	-	-	-	-
Cash year end	10.48	25.22	70.80	65.09	160.34

Investment Fundamentals	FY25a	FY26	FY27	FY28	FY29
Liquidity					
Current Ratio	7.9	10.2	32.8	38.5	94.5
Quick Ratio	2.2	2.0	2.1	1.9	1.9
Solvency					
Debt to Equity	0.0	0.0	0.0	0.0	0.0
Debt to Assets	0.0	0.0	0.0	0.0	0.0
LT Debt to Assets	0.0	0.0	0.0	0.0	0.0
Profitability					
Net Margin	n/a	n/a	77%	n/a	91%
ROA	-29%	-25%	50%	-8%	55%
ROE	-31%	-27%	51%	-8%	55%
Valuation					
P/E	n/a	n/a	5.8	n/a	2.6
P/B	8.7	6.2	2.9	3.2	1.4
EV/EBITDA	n/a	n/a	4.1	n/a	0.9
EV/Sales	n/a	n/a	3.4	n/a	0.8

Key Risks

Clinical development and regulatory risks

While the independently validated ACCENT data is encouraging, the trial remains a single-arm Phase 1b/2a study with 64 patients. The primary risk is that these results may not be replicated in a larger, randomised, placebo-controlled pivotal Phase 3 trial. Historically, many promising Phase 2 oncology assets have failed to demonstrate a statistically significant benefit in Phase 3 due to inherent differences in patient selection, trial design, and statistical powering. Metastatic pancreatic ductal adenocarcinoma (mPDAC) remains a notoriously difficult indication with a high rate of clinical trial failure, and the broader class of FAK inhibitors has a mixed clinical track record. The 11.1-month mOS, while commercially viable, does not yet demonstrate a statistically significant separation from the NALIRIFOX benchmark – a larger trial will be needed to confirm superiority or non-inferiority with a differentiated safety and response profile. Furthermore, like other ATP-competitive kinase inhibitors, narmafotinib only blocks FAK's enzymatic function, leaving the protein's kinase-independent scaffolding role intact, which may mediate adaptive resistance over time.

Competition and market risk

The mPDAC treatment landscape is intensely competitive and rapidly evolving. NALIRIFOX has established an 11.1-month mOS benchmark as the new standard of care, meaning narmafotinib's survival data alone – while matching this figure – does not yet demonstrate clear superiority. Direct competition from Verastem Oncology's defactinib, which is already FDA-approved for another indication and is being aggressively developed in combination for PDAC in the RAMP 205 trial, presents a potential first-mover advantage that could narrow narmafotinib's market opportunity if its combination is approved first. Additionally, several other companies with different mechanisms of action (including Arcus Biosciences' quemliclustat in Phase 3, and Cantargia's nadunolimab) have reported strong survival signals in mid-to-late-stage trials. The success of any of these competitors could further raise the efficacy bar for clinical adoption.

Commercialisation risk

Amplia's corporate strategy is predicated on securing a licensing partnership with a major pharmaceutical company to fund the substantial costs of Phase 2b/3 development and global commercialisation. The company's ability to negotiate favourable deal terms is contingent on the totality of its clinical data package, which now rests almost entirely on the mature ACCENT readout following the discontinuation of recruitment in AMPLICITY. While the independently validated CR signal and 11.1-month mOS strengthen Amplia's negotiating position, the loss of AMPLICITY removes a parallel data stream that could have supported deal value. Failure to secure a partner would place the full financial burden of a pivotal study directly on the company, likely necessitating substantial and highly dilutive capital raises. The timing and terms of any partnership remain uncertain and represent a key source of binary risk.

Funding and financial risks

Following the A$25M capital raise in July 2025, Amplia's cash position was guided to extend into 2027. With recruitment to the AMPLICITY trial now halted, the trial-cost component of the cash burn should reduce, modestly extending the effective runway; the March quarter Appendix 4C will provide a precise update. However, the existing cash balance is not sufficient to fund a pivotal Phase 2b/3 study of narmafotinib plus gem/Abraxane, which we estimate would require in excess of A$40M depending on final design. Amplia will therefore need to secure additional capital, whether through a strategic partnership, an equity raise, non-dilutive sources, or a combination, to fund a registrational programme to completion. A standalone equity raise, if required, would likely be dilutive and is dependent on prevailing market conditions, share price levels, and clinical progress at the time of issuance.

Asset concentration risk

Amplia's valuation and near-term prospects are almost entirely dependent on the clinical and commercial success of a single lead asset, narmafotinib, and within that, increasingly on a single trial: ACCENT and its planned Phase 2b/3 follow-on with gem/Abraxane. The discontinuation of recruitment in the AMPLICITY trial removes a parallel data stream and concentrates clinical risk further. Any clinical failures, safety issues, regulatory delays, or manufacturing problems related to narmafotinib would have a material and disproportionately adverse impact on the company's valuation.

Intellectual property risk

The long-term commercial viability of narmafotinib relies on Amplia's ability to successfully obtain, maintain, and defend its intellectual property portfolio. While the company has a multi-layered IP strategy with protection extending into the 2040s, competitors may challenge existing patents or pending applications. There is no guarantee that Amplia's current IP strategy will provide sufficient protection to prevent the entry of competing products.

Evolution Capital Ratings System

Recommendation Structure	• **Buy:** The stock is expected to generate a total return of >10% over a 12-month horizon. For stocks classified as 'Speculative', a total return of >30% is expected. • **Hold:** The stock is expected to generate a total return between -10% and +10% over a 12-month horizon. • **Sell:** The stock is expected to generate a total return of <-10% over a 12-month horizon.
Risk Qualifier	• **Speculative ('Spec'):** This qualifier is applied to stocks that bear significantly above-average risk. These can be pre-cash flow companies with nil or prospective operations, companies with only forecast cash flows, and/or those with a stressed balance sheet. Investments in these stocks may carry a high level of capital risk and the potential for material loss.
Other Ratings:	• **Under Review (UR):** The rating and price target have been temporarily suppressed due to market events or other short-term reasons to allow the analyst to more fully consider their view. • **Suspended (S):** Coverage of the stock has been suspended due to market events or other reasons that make coverage impracticable. The previous rating and price target should no longer be relied upon. • **Not Covered (NC):** Evolution Capital does not cover this company and provides no investment view.

Expected total return represents the upside or downside differential between the current share price and the price target, plus the expected next 12-month dividend yield for the company. Price targets are based on a 12-month time frame.

Disclaimer & Disclosures

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