EVOLUTION CAPITAL

ATX | UPDATE REPORT | HEALTHCARE

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Lilly Puts Its Lead RAS Asset Alongside Narmafotinib

Amplia Therapeutics Ltd.

Amplia has signed a Clinical Trial Collaboration and Supply Agreement (CTCSA) with Eli Lilly to run a Phase 1b/2b study of narmafotinib with olomorasib, Lilly's next-generation KRAS G12C inhibitor, in previously treated advanced non-small cell lung cancer (NSCLC), opening at Australian and US sites in late CY26. Two days later at the Bioshares Biotech Summit, management disclosed the trial architecture (~20 patients in dose escalation, ~40 in a randomised two-dose comparison) alongside a restated three-part pancreatic registration pathway and a September 2026 start for ACCENT-mini.

The Deal Structure

Amplia gets Olomorasib supplied in kind, Lilly's clinical input into protocol design (an advanced draft was jointly finalised before signing), and shared access to the dataset. There are no payments, options, commercial rights, and Lilly has no obligation to do anything further. The arrangement is non-exclusive (and Amplia themselves flag a second unnamed "KRASi combination" from 2027). Amplia sponsors, runs and funds the study. That is the material economic term. We expect cost in the range of A$8-12m across FY27-FY29.

The Bioshares deck disclosed the design, which had not been public at the time of the ASX release:

- **Phase 1b (~20 patients):** narmafotinib dose escalation at 200mg, 400mg then 600mg once daily, on a fixed backbone of olomorasib 100mg twice daily.

- **Phase 2b (~40 patients):** randomised head-to-head comparison of two narmafotinib doses on the same olomorasib backbone, following FDA *Project Optimus* dose-optimisation principles.

- **Population:** previously treated (second-line) KRAS G12C-mutant advanced or metastatic NSCLC. Sites in Australia and the US.

Narmafotinib will be dosed continuously once daily, consistent with the pivot away from ACCENT's intermittent 12-of-28-day schedule. That efficacy package was generated at roughly 43% theoretical dosing coverage, so continuous dosing is the route to a deeper effect. Our view is the lung program is real optionality, but with a real cash cost. Lilly's diligence is a credible external signal on an asset not yet validated by big pharma.

Valuation

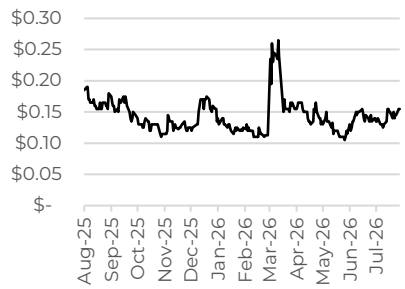
Fair value moves to A$0.49/sh, from A$0.48. We have rolled the model onto Amplia's FY26 result, pushed our assumed licensing schedule back twelve months so the first payment now falls in FY28, added A$10m of lung-trial cost across FY27-FY29, and moved our AUD/USD assumption to 0.70. A year of clinical progress and a materially stronger balance sheet are very nearly offset by a longer, more expensive and more currency-exposed path to the same milestones. The net movement is one cent, and we would caution against reading that as evidence little has changed. Recommendation unchanged at Speculative Buy. See Influences on Our Valuation.

Recommendation	**Spec Buy**
Previous Close	**$0.155**
Fair Valuation	**$0.49**
TSR	**216%**

Company Profile

Market Cap	$79.6M
Enterprise Value	$51.7M
SOI (undiluted)	513.3M
Free Float	89.2%
ADV (3-month)	$173.4k
52-Week Range	$0.10 - $0.275

Price Performance



%	1M	3M	12M
Absolute	6.9%	10.7%	-16.2%
ASX/S&P200	2.6%	6.6%	0.6%

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. Mature Phase 1b/2a ACCENT data in first-line metastatic pancreatic cancer showed a 35.9% response rate and 11.1-month median overall survival. With FDA Fast Track and Orphan Drug designations secured, a three-part registrational pathway now set out, and a clinical collaboration signed with Eli Lilly in KRAS G12C-mutant lung cancer, Amplia is positioned at a key value inflection point ahead of anticipated partnership discussions.

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A Reminder: What RAS Inhibitors Do

RAS proteins are molecular switches sitting just inside the cell membrane, cycling between an inactive GDP-bound state and an active GTP-bound state. When active, RAS turns on the RAF–MEK–ERK and PI3K–AKT–mTOR cascades that instruct a cell to grow and divide. Mutations at codons 12, 13 or 61 jam the switch "on", and the cell proliferates without an external growth instruction.

KRAS is the most frequently mutated oncogene in human cancer, present in roughly 90% of pancreatic ductal adenocarcinoma (PDAC) and 30–40% of lung adenocarcinoma and colorectal cancer. For four decades it was regarded as undruggable: a smooth protein surface with no obvious pocket, and picomolar affinity for GTP that ruled out competitive inhibition. That changed in 2013, when a cryptic "switch-II" pocket was identified adjacent to the cysteine created by the G12C mutation. This led to the development of the first two G12C inhibitors – sotorasib (Lumakras, Amgen, 2021) and adagrasib (Krazati, Mirati/BMS, 2022) – which reached the market for previously treated NSCLC.

Each produced a median progression-free survival (mPFS) of only 5.5–5.6 months in randomised second-line lung studies. Regardless, commercial uptake followed: 2025 sales were roughly US$363m for Lumakras and US$205m for Krazati. Responses are real but short-lived, because tumours reactivate signalling through parallel routes within months. The field's answer has been twofold: build better G12C inhibitors and stop using them alone. There are now 60-plus RAS-directed programmes in development, spanning G12C, G12D and pan-RAS "RAS(ON)" multi-selective agents that bind the active GTP-bound state directly.

Key Recent Developments

RASolute 302 (second line, pancreatic)
Revolution Medicines' daraxonrasib – a RAS(ON) multi-selective inhibitor blocking the active state of both mutant and wild-type RAS – reported topline results in April 2026 and full data at the ASCO plenary in June, published simultaneously in the New England Journal of Medicine. In 500 previously treated metastatic PDAC patients randomised 1:1 against investigator's choice chemotherapy, median overall survival (mOS) was 13.2 months versus 6.7 months (HR 0.40, $p < 0.0001$). In a disease where second-line therapy has historically delivered six months, that is the largest single advance in a generation. The NDA was accepted for FDA review in July 2026.

RASolute 303 (first line, pancreatic)
Dosing began in April 2026 in a ~900-patient, three-arm global Phase 3 randomising treatment-naïve metastatic PDAC 1:1:1 to daraxonrasib monotherapy (300mg); daraxonrasib 200mg plus gemcitabine/nab-paclitaxel for up to six months then daraxonrasib maintenance; or gemcitabine/nab-paclitaxel alone. Co-primary endpoints are PFS and OS, enrolling irrespective of RAS genotype.

RASolute 303 matters directly to Amplia. Its combination arm is structurally the same idea as ACCENT: a targeted agent layered onto the gemcitabine/nab-paclitaxel backbone in first-line metastatic disease. It will therefore answer, with randomised evidence and at scale, whether that architecture beats chemotherapy alone. It will also test whether the chemotherapy backbone is needed at all.

Olomorasib: Lilly's Flagship RAS Asset

Olomorasib (LY3537982) is an oral, second-generation covalent G12C inhibitor engineered for highly efficient covalent bond formation, achieving >90% sustained target occupancy at low systemic exposure. The design intent was combinability. Sotorasib and adagrasib achieve target coverage only at exposures that leave little tolerability headroom for a partner drug, which partly why first-generation combination attempts, particularly with immunotherapy, ran into hepatotoxicity. The data supports the design. In the Phase 1/2 LOXO-RAS-20001 study, olomorasib monotherapy in second-line NSCLC produced an mPFS of 8.1 months and a 41% response rate. With pembrolizumab in the first line, the response rate was 74% (91% disease control), rising to approximately 90% in PD-L1-high patients. Lilly is now running two global Phase 3 registrational studies: SUNRAY-01 in advanced NSCLC (n≈1,264) and SUNRAY-02 in resected or unresectable stage II–III disease (n≈700).

FAK & RAS Inhibition Together

Focal adhesion kinase (FAK) is a cell's connection to the scaffolding around it. Tumours sit inside a dense mesh of collagen and support cells known as the stroma, and FAK is the switch that reads signals from that mesh and converts them into instructions to survive, grow and move. It does this through its own set of pathways (PI3K–AKT–mTOR, Hippo/YAP, Wnt, RhoA) — and critically, those pathways run *alongside* the RAS–MAPK cascade rather than downstream of it. Switching off RAS does not switch off FAK.

That is the entire basis for combining the two. When a G12C inhibitor shuts down RAS–MAPK signalling, surviving tumour cells are not defenceless: the best-documented escape route is to turn FAK up, restoring survival signalling through the FAK–YAP axis. FAK activation also thickens the stroma itself, building a denser physical barrier around residual disease. Blocking FAK closes that escape route before the tumour can use it

Three independent lines of evidence now support this:

- **Preclinical:** The FAK–YAP synergy with G12C inhibition was formally described in *Advanced Science* in 2021 and has been replicated across multiple models. At the AACR Special Conference on RAS Oncogenesis in March 2026, Amplia presented its own data showing narmafotinib enhances KRAS inhibitor activity in pancreatic, lung and ovarian models.

- **Clinical.** In July 2026, InxMed published in The Lancet Respiratory Medicine results for its FAK inhibitor ifebemtinib plus the G12C inhibitor garsorasib in first-line KRAS G12C-mutant NSCLC in China. The confirmed response rate was 82% (27/33 enrolled; 87% of evaluable patients) in an all-oral, chemotherapy-free regimen, with no overlapping toxicity between the agents. The combination has China NMPA Breakthrough Therapy designation and a randomised Phase 3 underway.

- **Regulatory.** In May 2025 the FDA granted accelerated approval to Verastem's defactinib (a FAK inhibitor) with avutometinib in KRAS-mutant recurrent low-grade serous ovarian cancer. This was the first approval of a FAK inhibitor in any setting, and specifically as a combination partner in RAS-driven disease.

Our view is that the mechanistic case now rests on a stronger platform than that which we set out in April. Preclinical evidence is now backed up with a peer-reviewed clinical dataset.

Pipeline Overview

The pancreatic pathway remains the value core and is unchanged in substance: ACCENT-mini establishes the daily-dosing safety and PK package, that package informs an FDA meeting, and the registrational Phase 2b selects the dose for a pivotal Phase 3. What has changed is that the timeline now shows a "KRASi arm" appended to ACCENT-reg from 2028: signal of intent to bring a RAS combination into the pancreatic registration programme itself rather than run it separately. Given RASolute 302 and 303, that is the logical destination.

PRROSE is a small, investigator-initiated and ANZGOG-sponsored, so cash exposure is limited and endpoints are not registrational.

Figure 1: Amplia clinical pipeline as at August 2026. Source: Amplia Therapeutics, Bioshares Biotech Summit presentation, 6 August 2026; ASX releases.

Programme	Indication	Design	Status / Next Step
ACCENT	1L metastatic PDAC	Ph1b/2a, n=64 at 400mg, narmafotinib + gem/nab-paclitaxel	Complete. ORR 35.9%, CR 7.8%, DCR 70%, mPFS 7.7 mths, mOS 11.1 mths
ACCENT-mini	1L metastatic PDAC	n=12, two cohorts, daily dosing at two dose levels, 3–4 Australian sites; safety, PK, efficacy, biomarkers	First patient dosing Sept 2026; safety/PK review Q1 CY27
ACCENT Reg – P2b	1L metastatic PDAC	Head-to-head comparison of two daily doses (Project Optimus)	FDA meeting Q2 CY27; start 2H CY27
ACCENT Reg – P3	1L metastatic PDAC	Pivotal registrational study at the P2b-selected dose	Design TBC; supports FDA submission
PRROSE	High-grade serous ovarian	Investigator-initiated, ~15–20 patients, narmafotinib + carboplatin/paclitaxel pre-interval debulking; endpoints safety, surgical eligibility, biomarkers	ANZGOG-sponsored, led by Dr Gwo Yaw Ho (Monash); recruiting from 2H CY26
NSCLC (Lilly)	2L KRAS G12C NSCLC	Ph1b/2b, ~60 patients, narmafotinib + olomorasib, Aus + US	Protocol finalisation; planned start late CY26

Influences on Our Valuation

We lift our risked fair value one cent to A$0.49/sh, from A$0.48, and retain our Speculative Buy recommendation. The valuation remains a sum-of-the-parts of US and EU first-line metastatic PDAC only, risked at an 18% Phase 2-to-approval probability of success (PoS), plus net cash and less present-valued corporate overhead. Neither NSCLC nor ovarian carries any value today.

That single cent conceals considerably more movement beneath it. Four changes account for the difference, and they very nearly cancel:

- **Rolling the model onto the FY26 result adds approximately six cents.** Amplia closed 31 March 2026 with A$27.9m of cash, or A$32.3m including the R&D incentive receivable and net of lease liabilities. At June 30, ATX reported $23.98m cash. The balance sheet is a year stronger, our diluted share count falls to 615.4m from 620.5m on the updated option position, and every forecast cash flow is discounted one year less.

- **Deferring the licensing schedule and funding the lung study costs approximately one cent.** We now assume the first payment, a US$50m upfront, lands in FY28 rather than FY27, with each subsequent milestone pushed back a year in step. Nothing in the CTCSA or the company's guidance dates a partnering event; the deferral reflects our own read that a registrational-stage deal is more likely to follow the ACCENT-mini safety and PK package and the Q2 CY27 FDA meeting than to precede them. We have separately added A$10m of narmafotinib spend across FY27-FY29 for the lung study, phased A$2m, A$4m and A$4m, within our A$8-12m estimate.

- **Moving our AUD/USD assumption to 0.70 from 0.66 costs approximately three cents.** This is the largest single drag on the valuation. Every royalty and milestone in the model is struck in US dollars and converted at a constant rate, so a stronger Australian dollar reduces the Australian-dollar value of identical US receipts by 5.7%. Readers should treat this as a lever rather than a forecast: we hold the rate flat across a horizon extending to FY37, and the valuation is more sensitive to it than to any assumption we would describe as a view.

- **Refinements to our corporate cost assumptions account for the balance.** Corporate overhead is now carried on a consistent perpetuity basis with programme cash flows.

The composition matters more than the net. A year of realised progress has been almost entirely offset by a longer, costlier and more currency-exposed path to the same milestones. The unchanged inputs are as important as the changed ones: PoS remains 18%, the discount rate remains 14%, and our first-line mPDAC commercial assumptions are unrevised.

The deferral sharpens the funding question rather than creating it. On our revised numbers Amplia exits FY27 with approximately A$17.9m and receives no licensing income until FY28. Our model already assumed the pancreatic registrational programme requires a partner or a raise; a second sponsored trial brings that decision forward and raises the probability any raise occurs before rather than after a partnering event. We have lifted near-term opex accordingly, but we have deliberately not yet modelled the dilution itself. Our fair value therefore assumes a funding path the balance sheet alone does not support, and an equity raise struck near the current price would be materially dilutive to it. We regard this as the most significant unmodelled item in the valuation.

Our April note identified single-asset, single-trial concentration as a core risk, and narmafotinib now has three independent shots on goal. Partnering leverage: a FAK asset with a combination franchise spanning chemotherapy and targeted therapy is a better licensing proposition than one tied to a single chemotherapy backbone, bearing on both deal probability and terms. Cross-programme read-through: a positive lung result would raise the prior on FAK as a general-purpose sensitiser, feeding back into the pancreatic PoS rather than sitting in isolation.

RASolute 303 will determine whether daraxonrasib compresses narmafotinib's first-line pancreatic niche or creates a combination partner for it; its daraxonrasib-plus-gemcitabine/nab-paclitaxel arm directly overlaps ACCENT's architecture. We are not willing to resolve that in the model yet. Separately, our first-line mPDAC commercial assumptions were set before the RAS class demonstrated a 13.2-month mOS in the second line and would require revisiting upon announcement of RASolute 303 data.

We expect to carry lung as a separately risked rNPV at a low single-digit PoS – early-phase, ~60 patients, non-randomised – sized on the KRAS G12C-mutant second-line population (~13% of NSCLC), not the whole indication. On preliminary work the risked contribution is unlikely to exceed the incremental trial cost near-term: the value is in optionality and the partnering signal, not modelled cash flows.

Financial Summary

VALUATION DETAILS	
Share Price (A$)	$0.155
Market Cap (A$m)	$79.6
Enterprise Value (A$m)	$51.7
Fair Value/Share (A$)	**$0.49**

FINANCIAL STATEMENTS (A$m)	FY25A	FY26A	FY27E	FY28E	FY29E
Income Statement					
Revenue	$4.1	$5.5	$5.2	$16.7	$16.7
EBITDA	($6.4)	($7.6)	($10.8)	$1.3	$1.3
EBIT	($6.5)	($7.7)	($10.8)	$1.3	$1.3
Net Income	($6.6)	($7.7)	($10.8)	$0.9	$0.9
Balance Sheet					
Cash & Cash Equivalents	$10.9	$27.9	$17.9	$78.7	$67.7
Inventory	-	-	-	-	-
Receivables	$3.8	$4.8	$5.2	$4.8	$4.8
Other Assets	$8.3	$8.8	$8.8	$8.8	$8.8
Total Assets	**$22.9**	**$41.4**	**$31.9**	**$92.3**	**$81.3**
Total Debt	-	-	-	-	-
Other liabilities (incl. deferred rev)	$1.9	$2.0	$3.2	$62.7	$50.8
Total Liabilities	**$1.9**	**$2.0**	**$3.2**	**$62.7**	**$50.8**
Shareholders' Equity	**$21.0**	**$39.5**	**$28.7**	**$29.6**	**$30.5**
Cash Flow Statement					
Net Income	($6.6)	($7.7)	($10.8)	$0.9	$0.9
Add: D&A	$0.1	$0.1	-	-	-
Less: ΔNWC	-	($1.1)	-	-	-
Cash Flow from Ops.	**($6.5)**	**($8.7)**	**($10.8)**	**$0.9**	**$0.9**
Cash Flow from Inv.	-	($0.1)	-	-	-
Equity Raised (net)	$17.3	$25.9	-	-	-
Less: Dividends Paid	-	-	-	-	-
Cash Flow from Fin.	**$17.3**	**$25.9**	**-**	**-**	**-**
UFCF	**($6.5)**	**($8.8)**	**($10.8)**	**$0.9**	**$0.9**

PER SHARE DATA	FY25A	FY26A	FY27E	FY28E	FY29E
Diluted Shares Out (m)	499.2	615.4	615.4	615.4	615.4
EPS (A$)	($0.013)	($0.014)	($0.018)	$0.001	$0.001
Dividends per Share (A$)	$0.000	$0.000	$0.000	$0.000	$0.000
Payout	0.0%	0.0%	0.0%	0.0%	0.0%
Franking	0.0%	0.0%	0.0%	0.0%	0.0%

RATIOS	FY25A	FY26A	FY27E	FY28E	FY29E
Liquidity					
Current Ratio	7.91x	19.78x	8.00x	29.56x	25.69x
Quick Ratio	7.75x	19.54x	7.86x	29.41x	25.54x
Solvency					
Debt to Equity	0.00x	0.00x	0.00x	0.00x	0.00x
Equity to Assets	91.7%	95.2%	89.8%	32.1%	37.6%
Profitability					
ROA (Return on Assets)	-28.7%	-18.6%	-33.7%	1.0%	1.1%
ROE (Return on Equity)	-31.3%	-19.5%	-37.5%	3.1%	3.0%
EBITDA Margin	-158.4%	-138.4%	-206.4%	7.9%	7.9%
NPAT Margin	-161.9%	-140.4%	-206.4%	5.5%	5.5%
Growth					
Revenue	n/a	35.2%	(4.9%)	219.7%	0.0%
EBITDA	n/a	n/m	n/m	n/m	0.0%
Underlying NPAT	n/a	n/m	n/m	n/m	0.0%
EPS	n/a	n/m	n/m	n/m	0.0%
Valuation					
P/E	n/m	n/m	n/m	103.9x	103.9x
EV/Revenue	12.7x	9.4x	9.9x	3.1x	3.1x
EV/EBITDA	n/m	n/m	n/m	39.4x	39.4x
Dividend Yield	0.0%	0.0%	0.0%	0.0%	0.0%

Key Risks

Clinical development risk
ACCENT is a single-arm, 64-patient Phase 1b/2a study and its efficacy signal may not replicate in a randomised, controlled trial. The 11.1-month mOS does not separate significantly from the NALIRIFOX benchmark, and inverts to non-significance when tested against the contemporary NAPOLI-3 control arm. The FAK class has a mixed clinical record, and narmafotinib, like other ATP-competitive inhibitors, leaves FAK's kinase-independent scaffolding role intact, a plausible route to adaptive resistance. The AMPLICITY halt demonstrated that the chemotherapy backbone itself can constrain a combination.

Regulatory and execution risk
The registrational pathway is set out but not agreed in final form. ACCENT-mini (n=12) must deliver the daily-dosing safety and PK package, that package must satisfy the Q2 CY27 FDA meeting, and the Phase 2b must then select a dose under Project Optimus. Each step can slip or change the design. Recruitment in mPDAC is competitive, and running two programmes across Australian and US sites adds execution risk to both.

Competition and market risk
NALIRIFOX's 11.1-month mOS is the established standard of care, so narmafotinib's survival data alone does not demonstrate superiority. Verastem's defactinib is already FDA-approved elsewhere and is in RAMP 205 in PDAC, and may take first-mover advantage in FAK. Revolution Medicines' daraxonrasib delivered 13.2 months mOS in the second line and is now in first-line Phase 3 with an architecture that directly overlaps ACCENT's. Arcus, Immuneering and others may raise the bar further, and competitor readouts in 2027 may precede ACCENT's successor study.

Collaboration risk
The Lilly CTCSA carries no payments, options or commercial rights, and Lilly has no obligation beyond supplying olomorasib. The arrangement is non-exclusive and can end without compensation, and Lilly's own SUNRAY programme has first call on the asset. Amplia sponsors, funds and bears the full execution risk of the study while holding none of the partner economics.

Partnering and funding risk
Strategy depends on a licensing partnership to co-fund a pivotal study we estimate at more than A$40m. On our numbers Amplia exits FY27 with approximately A$17.9m and receives no licensing income until FY28. Our fair value therefore assumes a funding path the balance sheet alone does not support, and we have not modelled the dilution; a raise struck near the current price would be materially dilutive to it. The timing and terms of any partnership remain the single largest binary risk to the investment case.

Licensing terms are our assumption, not an agreement
No licensing agreement exists. The US$50m upfront we now place in FY28, the milestones that follow it and the 12.5-15% royalty are Evolution Capital estimates drawn from precedent transactions, not company guidance. A deal concluded later, on worse terms, or not at all would change the valuation materially, and this schedule is the single largest driver of the near-term cash profile in our model.

Currency risk
Every royalty and milestone in our valuation is denominated in US dollars and converted at a constant AUD/USD rate of 0.70, held flat to FY37. A four-cent move in the currency is worth roughly three cents of fair value, which makes AUD/USD a larger swing factor than several of the clinical assumptions a reader would reasonably consider more consequential.

Valuation methodology and sensitivity
Our fair value is a risk-adjusted NPV, and small changes in unobservable inputs move it a long way. At an unchanged 14% discount rate, a 10% probability of success rather than 18% reduces fair value to approximately A$0.28; holding PoS at 18% and lifting the discount rate to 16% gives approximately A$0.39. Around half of gross programme value sits in a terminal value that assumes royalties persist and grow beyond narmafotinib's patent protection. Benchmarking against MPACT and NAPOLI-3 is a cross-trial comparison, not randomised evidence.

Asset concentration and intellectual property risk
Value is almost entirely dependent on a single molecule and, within that, largely on a single trial pathway. The AMPLICITY discontinuation removed a parallel data stream and concentrates clinical risk further. Any setback, safety signal, regulatory delay or manufacturing issue would have a disproportionate share price impact. Long-term viability also depends on defending a patent estate that extends into the 2040s but may be challenged.

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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