EVOLUTION CAPITAL

ASX:SNT
Healthcare
Update Report

Tuesday, 17 June 2025

A Win on Symptoms, A Wait on SVR

Evolution Capital provides an update on Syntara ('SNT'), maintaining a speculative Buy rating and reaffirming our price target of $0.235. Syntara has released further interim data from its ongoing Phase 2 trial of SNT-5505 in combination with ruxolitinib for the treatment of myelofibrosis (MF). The update provides the first 52-week dataset and offers a clearer picture of both durability and clinical relevance. We believe the results reinforce the drug's clinical potential and support continued advancement.

Is SNT-5505 cracking the myelofibrosis code?

The June interim cut takes us from 390 to 499 patient-weeks of exposure and delivers the first 52-week read-out: 73 % of evaluable patients hit TSS50 by Week 24, driving and mean symptom relief to -63 % out at Week 52. That is double the 30-40% response rates seen with competing add-ons and comes in a cohort battered by a median 38 months on ruxolitinib (standard of care). Importantly, haematology remains flat (one transfusion-dependent patient now ≥50% transfusion-free; platelets steady) and, across 499 patient-weeks, zero treatment-related serious adverse events (SAEs) have emerged. In short, the drug is hitting the inflammation switch hard without tipping patients into cytopenia-driven toxicity. This suggests a profile tailor-made for chronic use.

Why the modest spleen response and does it matter?

Headline SVR35 (a ≥ 35% reduction in spleen volume) sits at 1/9 by Week 24 and 3/9 at beyond 24 weeks, prompting inevitable whispers of "sub-par efficacy." Context is everything: (i) these patients started with years-old splenomegaly where fibrotic scaffolding is far harder to reverse (pelabresib in an equivalent phase 2, for example, saw 68% SVR35 at 24 weeks but where patients had no prior ruxolitinib treatment); (ii) volumetric thresholds are binary – a single extra responder would have lifted the rate to 44%; and (iii) 78% of patients still showed stable or shrinking spleens without ruxolitinib dose escalation. Historically, durable symptom control has preceded structural spleen changes in late-line MF studies. If this pattern holds, incremental SVR wins should emerge as more patients clear the 52-week mark.

Our read:

Symptom wins are real, spleen data needs time, and the unblemished safety bar underpins a clean transition into a pivotal combination study. We reaffirm our A$0.235 per share valuation: the risk-reward skew stays intact while the maturing dataset inches SNT-5505 closer to the all-important topline data readout in H2 this calendar year.

Recommendation	**SPEC Buy**
Previous Close	**$0.057**
Target Price	**$0.235**

Company Profile

Market Cap	$92.6m
SOI (undiluted)	1.625bn
Free Float	~86%
ADV (3-month)	~$277k
52-Week Range	$0.022 - $0.095

Price Performance

Company Overview

Syntara Limited (ASX: SNT) is a clinical-stage drug developer with in-house drug discovery expertise, focused on innovative treatments for blood cancers, inflammation, and fibrosis. Its lead candidate, SNT-5505, a pan-LOX inhibitor, is showing promising results in myelofibrosis trials. The company is also advancing therapies for MDS, neuroinflammation, and skin scarring in collaboration with leading institutions. Listed on the ASX, Syntara is pioneering novel solutions for high unmet medical needs.

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Click here to access Evolution Capital's Initiation Report of SNT published 27 March 2025.

Further Catalysts	Timeline
Initiation of Phase 1c/2 trial for SNT-5505 in low/intermediate-risk MDS	H1 2025
Initiation of Phase 1c/2 trial for SNT-5505 in high-risk MDS (AZALOX expansion phase)	H1 2025
FDA feedback request for next stage of SNT-5505 clinical development in MF	Q2 2025
Final Phase 2a data for SNT-5505 in MF	H2 2025
Phase 1c data for SNT-5505 in Myelodysplastic Syndrome (MDS)	H2 2025
Phase 2 trial results for SNT-4728 in Parkinson's Disease	H2 2025
Initiation of clinical trials for SNT-6302 in keloid scars & SNT-9465 in hypertrophic scars	H1 2025
Data from Phase 1a/b trial of SNT-9465	H1 2026

Interim to Interim Data Comparison

Data maturity: the dataset is now deeper. Since the cut-off of 14 Nov 2024 for the December interims, when 8/16 patients had reached 24 weeks and 5/16 had reached 38 weeks, the study has advanced to 11/16 patients at 24 weeks, 8/16 at 38 weeks and, for the first time, 5/16 completing the full 52-week course. Total drug exposure has grown from 390 to 499 patient-weeks and median follow-up from 24 → 36 weeks, giving a clearer view of durability.

Spleen Volume Reduction (SVR): clinically meaningful spleen responses edged forward but remain modest. SVR25 (spleen volume reduction ≥ 25%) has improved from 30 % (3/10) to 44 % (4/9) and SVR35 from 20 % (2/10) to roughly 33 % (3/9) as more patients crossed the ≥35 % volumetric threshold with longer dosing; 78 % (7/9) continue to show at least a stable spleen with no RUX dose escalation. The low absolute numbers mean each additional responder will move the headline rate materially. The effect of this is made evident when we timestamp SVR. Only 1 of 9 patients achieved SVR35 by week 24, and only 3/9 at any point beyond week 24.

Total Symptom Score (TSS): symptom control is the clear bright spot. The proportion achieving TSS50 has risen from 46 % at 12 weeks and 80 % at 38 weeks in December to 73 % already by 24 weeks in June, with durable mean reductions of 56 % at 38 weeks and 63 % at 52 weeks across the maturing cohort.

Safety & tolerability: this is looking good. Across both cut-offs there have been no treatment-related serious adverse events, most TEAEs remain Grade ≤ 2, and Grade 3/4 cytopenias are infrequent. Haemoglobin and platelet trends are flat, with one of two transfusion-dependent patients now showing a ≥50 % reduction in transfusion need and a transfusion-independent patient gaining 10 g/L in Hb.

Figure 1: Comparison of Key Clinical Metrics Between December 2024 and June 2025 Interim Data Cuts for the SNT-5505 + Ruxolitinib Phase 2 Study.

	December 2024	June 2025	What's Changed
Dataset	13 pts ≥ 12w 8 pts ≥ 24w 5 pts ≥ 38w	11 pts ≥ 24w 8 pts ≥ 38w 5 pts ≥ 52w 3 more still dosing	First 52w data now available; 24w pool is larger.
TSS50	6/13 (46%) at 12w 2/8 (25%) at 24w 4/5 (80%) at 38w	4/11 (36%) at 24w 8/11 (73%) at 24w+	Higher response rate at 24 wk and first durability read-out beyond 9 months
SVR25	3/10 (30%) anytime 1/10 (10% at 12w 2/10 (20%) at 24w	3/9 (33%) at 24w 4/9 (44%) at 24w+	Clinically meaningful SVR25 has risen but overall, spleen control maintained.
SVR35	1/10 (10%) at 12w a further 1/8 (20%) at 38w	1/9 (11%) at 24w 3/9 (33%) at 24w+	
Stable/reduced spleen	9/11 (82%)	7/9 (78%)	
Haematology	Hb & platelets generally stable; 1 of 2 transfusion-dependent (TD) pts saw ~7-% drop in transfusions.	Hb & platelets still stable; 1 of 2 TD pts now ≥50% drop; 1 of 7 pts gained 10g/L Hb.	Haematology remains steady.
Safety	No treatment-related SAEs	No treatment-related SAEs	Safety profile unchanged

Source: company data.

Phase 2 Trial Overview

The ongoing Phase 2 study of SNT-5505 is designed as a single-arm, open-label clinical trial assessing the drug's potential as an add-on therapy to ruxolitinib (rux), the current standard of care in myelofibrosis (MF). Specifically, it enrols patients who, despite extended prior treatment with RUX (median ~2 years), have reached a therapeutic plateau, meaning their symptoms or spleen volumes have ceased improving or begun deteriorating. Patients remain on their stable RUX dose, with SNT-5505 added on at a fixed, oral daily regimen. The primary endpoints of the study are standard efficacy measures, particularly spleen volume reduction (SVR25 and SVR35) and Total Symptom

Score (TSS50), alongside comprehensive safety and tolerability assessments. Secondary objectives include monitoring haematological stability, transfusion independence or reductions, and exploring biomarkers linked to disease response. The ultimate goal of this trial is to establish preliminary proof-of-concept data supporting SNT-5505's progression into pivotal studies as an effective, well-tolerated option addressing unmet clinical needs in MF patients inadequately managed by RUX alone.

Results Analysis

Total Symptom Score – The Clear Early Win

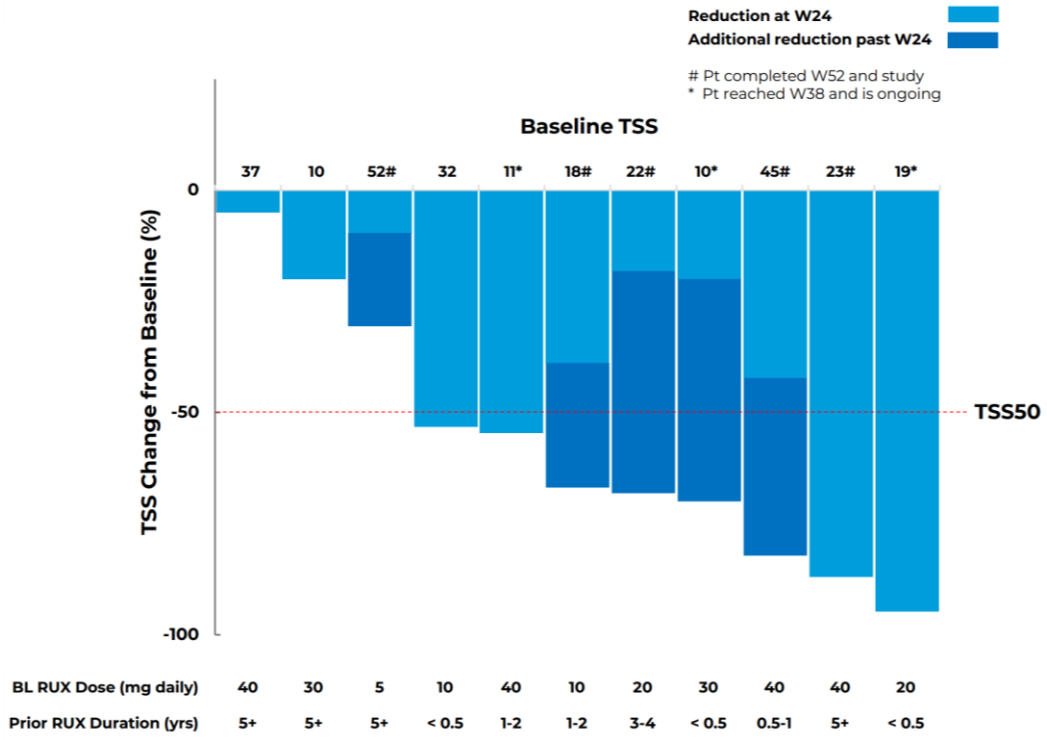
Understandable scepticism surrounds symptom-based endpoints, especially in early-stage clinical trials with limited patient numbers. As we know, the patients on trial had already reached therapeutic plateau prior to being dosed with SNT-5505: Syntara was facing an uphill battle from the get-go. However, both interim data readouts have addressed these concerns, with patient's exhibiting rapid and substantial symptom relief shortly after treatment initiation.

In last week's readout, 73% (8 of 11 evaluable patients) achieved at least a 50% reduction in their total symptom score (TSS50) by Week 24 or beyond. Even more compelling is the durability and progressive nature of these symptom improvements: the mean TSS reduction increased from 56% at Week 38 (n=8) to 63% at Week 52 (n=5). The mean absolute symptom reduction from baseline was approximately 6 points (median −39%, range from −5% to −95%). The data indicates ongoing improvements beyond the initial 24-week milestone, suggesting continued therapeutic benefit with extended treatment duration. This trend is critical, given that the patients enrolled had a high disease burden at baseline, with a median TSS score of 23 (range 10–52), reflecting severe symptomatic disease despite an average of three years on ruxolitinib.

Contextually, these symptom score outcomes position SNT-5505 very favourably relative to existing and late-stage developing treatments for MF. Historical data from comparable trials with alternative therapies typically report TSS50 response rates in the 30-40% range for patients similarly sub optimally managed on rux. Notably, these competitor benchmarks typically reflect patients earlier in their therapeutic journey, often with less prior exposure to ruxolitinib, meaning the hurdle for symptom relief was lower than for Syntara's heavily pre-treated cohort. In this light, achieving a TSS50 rate exceeding 70% represents a noteworthy clinical success and differentiating factor for SNT-5505.

Ultimately, the robust symptom improvements observed not only enhance patient quality-of-life but also substantiate SNT-5505's mechanism of action and therapeutic potential. These data are likely to strengthen the company's position in upcoming regulatory discussions and serve as a strong foundation for designing a pivotal Phase 2c/3 study.

Figure 2: Waterfall plot displaying percentage change from baseline in Total Symptom Score (TSS) by patient. TSS50 (≥50% reduction) is indicated by the dashed red line.



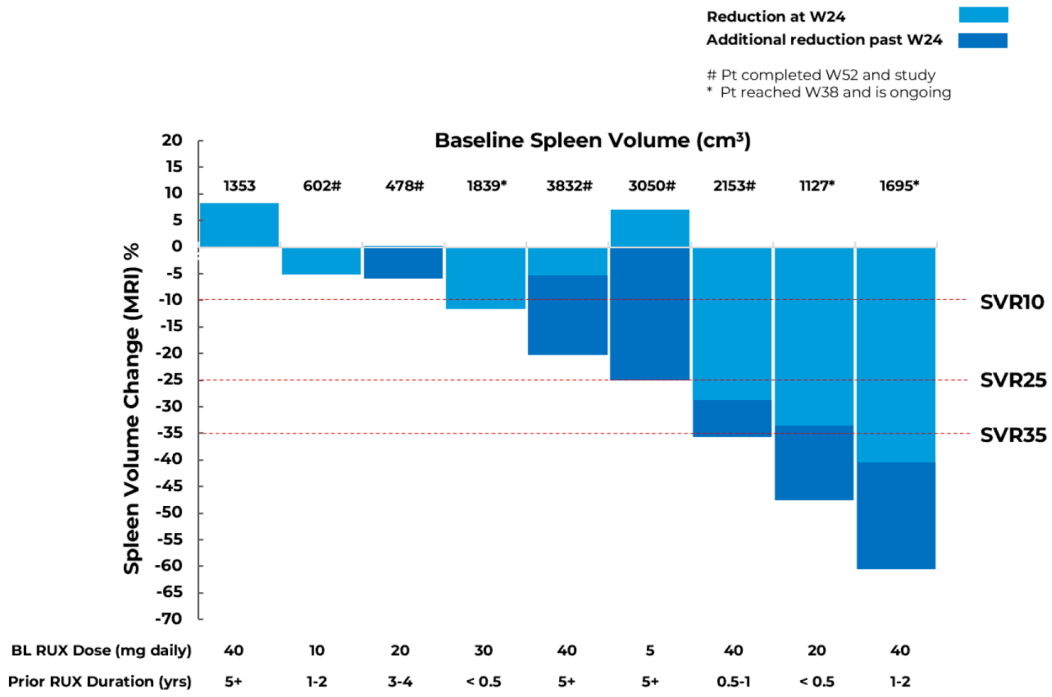
Source: company presentation.

Spleen Volume Reduction – Should We Be Worried?

Spleen volume reduction (SVR) is widely considered the most critical quantitative endpoint for evaluating drug efficacy in MF trials. This importance stems from the core pathology of MF itself: progressive fibrosis within the bone marrow forces blood cell production to shift to alternative sites, notably the spleen – a phenomenon known as extramedullary haematopoiesis. As a result, the spleen becomes severely enlarged (splenomegaly), causing debilitating symptoms such as abdominal discomfort, early satiety, and profound fatigue. Crucially, spleen enlargement also directly reflects the intensity of underlying disease processes, including fibrosis progression and abnormal blood cell proliferation. Therefore, achieving significant SVR is both clinically meaningful for patients and indicative of genuine disease modification rather than just symptom management.

The latest interims reported modest improvement in SVR outcomes, but the results leave a little to be desired. As outlined above, SVR25 data looks strong and 78% of patients exhibited at least stable spleen volumes. However, the critical SVR35 milestone, widely accepted as the regulatory benchmark for approval, was reached by only 1 of 9 patients by week 24, and just 3 of 9 at any time beyond week 24.

Figure 3: Waterfall plot showing individual patient spleen volume change (%) from baseline, as measured by MRI. All patients were on stable ruxolitinib dosing, and the majority had prior RUX exposure of ≥5 years.



Source: company presentation.

How does SNT-5505 compare to the rest? Before answering this question, we note that the SVR data must be interpreted within the critical context of patient populations and prior ruxolitinib exposure duration. In Syntara's Phase 2 combination study, patients had received ruxolitinib for a median of 38 months (range 5-89 months) prior to SNT-5505 initiation, representing an average of three years of prior JAK inhibitor therapy. As outlined in our initiation of coverage, approximately half of MF patients discontinue rux within 3 years and 75% within 5 years due to treatment failure or disease progression: therapeutic effect reduces with extended JAK inhibitor treatment. Moreover, prolonged exposure increases risks of serious adverse events including thrombosis, cardiovascular events, infections, and potential malignancies. Median survival is only ~15 months post JAK inhibitor discontinuation.

Among ruxolitinib-combination trials, pelabresib plus ruxolitinib has demonstrated the strongest SVR35 performance to date, achieving 65.9% versus 35.2% for placebo plus ruxolitinib in the pivotal phase 3 MANIFEST-2 study. This impressive result was presaged by equally robust data from the phase 2 MANIFEST study, where SVR35 reached 68% at week 24. Critically, both studies enrolled JAK inhibitor-naïve patients with no prior ruxolitinib exposure, representing an optimal treatment population. Similarly, in the frontline setting, historical ruxolitinib monotherapy trials like COMFORT-I demonstrated SVR35 rates of approximately 42% at week 24 in treatment-naïve patients. What must be noted in these trials is that they prevalence of adverse events was quite high, with serious anaemia common, suggesting that, regardless of incomparable characteristics of patient cohorts, the better spleen volume reduction is offset by poor tolerability. Another to add to this list is navtemadlin, where in its phase 2 trial 32% of those on-drug achieved SVR35 at week-24. However, median prior rux duration was 21.6 months and the tolerability profile was poor.

In contrast, studies evaluating drugs after ruxolitinib failure or in ruxolitinib-experienced populations show markedly lower SVR35 rates. Comparative Pacritinib trials in ruxolitinib-experienced patients showed SVR35 rates of approximately 19%. Navitoclax added to ongoing ruxolitinib achieved 26.5% SVR35 at week 24, but patients had a median prior ruxolitinib exposure of 82 weeks (approximately 1.6 years), considerably shorter than SNT-5505's patient population.

SNT-5505's SVR35 performance, while appearing modest at face value, becomes more clinically meaningful when contextualized against this backdrop of extensive prior ruxolitinib exposure.

SVR and TSS not correlating – why?

The interim data shows strong early symptom relief but only modest spleen volume reduction, highlighting a gap between the two main efficacy endpoints. This disconnect is not unique to SNT-5505, but understanding the underlying reasons is crucial for interpreting the clinical profile and future potential of the drug.

Why TSS Can Look Better Early:

- Fast-Acting Pathway: MF symptoms such as fatigue, night sweats, bone pain, and pruritus are largely driven by inflammatory cytokines, often mediated through the JAK/STAT pathway. SNT-5505's mechanism—LSD1 inhibition—appears to rapidly down-regulate these cytokines, leading to quick symptom relief.

- Sensitive, Continuous Measure: The MFSFA v4.0 diary used to assess TSS captures incremental, daily changes in symptoms, making it easier to detect and quantify improvements from various baseline levels. A 50% reduction is achievable from many different starting points, enhancing the sensitivity of this endpoint.

- More Evaluable Patients: Every patient with a baseline symptom diary is "evaluable" for TSS, so a larger proportion of the study cohort can contribute to this endpoint, reducing statistical noise and increasing the robustness of the signal.

- Inflammation vs. Fibrosis: Drugs like SNT-5505 can suppress cytokine-driven symptom flares without yet reversing the underlying fibrosis. This biological decoupling – rapid symptom relief without immediate structural organ change – has been observed in other MF trials as well.

Why SVR Can Lag:

- Slow-Moving Anatomy: SVR is a structural endpoint. Shrinking the spleen requires not only suppressing the malignant clone but also remodelling fibrotic tissue – a process that can take many months. Even by the June interim, only a minority of patients had reached 52 weeks of therapy, limiting the window for significant anatomical change.

- Binary, High Bar: SVR is a categorical endpoint – patients must cross a 25% or 35% volumetric threshold on MRI/CT to be counted as responders. A patient whose spleen shrinks by 20% (and who may feel much better) is still classified as a "non-responder" for SVR, underestimating the clinical benefit.

- Fewer Evaluable Patients: Only patients with a baseline spleen volume >450 cm^3 and complete imaging qualify for SVR analysis. In the June dataset, this was just nine patients. Small changes in responder numbers can disproportionately affect reported percentages.

The observed disconnect between rapid symptom improvement (TSS) and slower, more modest spleen volume responses (SVR) is a function of both the underlying disease biology and the clinical trial methodology. Symptom relief can be achieved quickly by modulating inflammatory pathways, while reversal of splenic fibrosis and anatomical remodelling is inherently slower and more challenging to measure, especially in small, early-phase cohorts.

The slower evolution of spleen response should not be interpreted as a lack of disease-modifying potential. As the trial matures, and more patients reach the 52-week mark, SVR rates are likely to improve, better reflecting the full therapeutic impact of the drug.

Valuation

Development & Commercialisation Assumptions

Our valuation remains unchanged: Speculative Buy with a price target of $0.235. We don't have any reason to change our assumptions around Syntara's development strategy. We expect the Company to complete the phase 2 trial of SNT-5505 in MF before advancing to a pivotal phase 3. And upon completion of this phase 3, we expect Syntara to secure a licensing deal with a major pharmaceutical partner.

Our assumptions regarding the timeline and costs associated with the Phase 3 clinical trial remain unchanged. The pivotal trial is expected to require approximately three years from initial patient enrolment to primary completion, followed by additional time for regulatory submission processes. We forecast the trial's overall duration, from initiation to NDA filing, to span approximately four years, beginning in early 2026 and concluding around early 2030.

We assume that this licensing deal will include an upfront payment of approximately US$150m, recognising zero further development risk, accompanied by up to US$400m in milestone payments, and a base 10% royalty. Given recent precedents, namely Takeda's deal with Keros for Elritercept (a phase 2 asset for MF and MDS) in 2024, commanding US$200m upfront and up to $1.1 billion in further payments, our expectation remains conservative.

Moreover, we maintain a conservative stance regarding market penetration rates for SNT-5505 in MF, reflecting historical adoption patterns and the competitive landscape. SNT-5505 will likely be commercialised initially as a second-line treatment for MF in those sub optimally treated previously with standard of care. In the US, initial market penetration is projected at 5% in FY31 (the first sales year post anticipated FDA approval in mid-2030), progressively rising to 20% by FY34. For global markets, slower initial adoption is expected, starting at 3% in FY31 and gradually increasing to 12% by FY34. Our pricing assumptions remain constant.

As for SNT's entry into the MDS market, we forecast Phase 3 completion post-FY29 with FDA approval expected in early-to-mid FY32 and ex-US launches by FY33. Uptake is expected to be slower than in MF due to treatment complexity and market caution, with US penetration starting at 1.5% in FY32 and reaching 6% by FY34, while ex-US adoption reaches 4% by FY35. Pricing assumptions are set at US$50,000 per patient per year in ex-US markets, consistent with benchmarks for haematology drugs. Despite a projected decline in incidence, MDS prevalence is expected to remain high, supporting long-term commercial potential.

SOTP Valuation

We apply a 13.3% probability of success (PoS) to our risk-adjusted net present value (rNPV) model for SNT-5505. This figure is derived from the midpoint between success rates for oncology and all ex-oncology orphan drug development programs, reflecting the dual nature of myelofibrosis (MF) and myelodysplastic syndromes (MDS) as both blood cancers and rare haematologic conditions. Specifically, PoS from Phase 1 to approval for oncology orphan drugs is 2.8%, while all ex-oncology orphan drug programs show a PoS of 23.8%. Taking the average of these two endpoints results in our conservative 13.3% PoS assumption, which appropriately captures the clinical and regulatory risk inherent in late-stage haematology drug development.

Our fair valuation for Syntara is based on a focused assessment of its flagship asset SNT-5505. We constructed risk-adjusted NPV models for MF and MDS respectively, combining together to form a "sum-of-the-parts" valuation.

Figure 4: SOTP and fair valuation summary tables.

SOTP	
Sum of PVs	597.52
Terminal Value (TV)	8,843.61
PV of (TV)	2,269.37
NPV of Program	2,866.89
PoS	13.3%
rNPV	**A$381.30m**

Valuation	
Net Debt	-17.91
Enterprise Value	363.39
Equity Value	381.30
Shares Outstanding (millions)	1,625.0
Fair Valuation	**A$0.235**

Source: Evolution analysis

Financial Statements

Income Statement *A$'000s*	FY23a	FY24a	FY25e	FY26e	FY27e
Revenue	-	-	-	-	-
Other Income	6.35	5.85	5.63	11.79	14.76
Total Revenue	**6.35**	**5.85**	**5.63**	**11.79**	**14.76**
Operating expenses	-17.71	-18.90	-18.17	-38.07	-47.67
EBITDA	-11.36	-13.05	-12.54	-26.28	-32.91
D&A	-1.85	-0.23	-	-	-
EBIT	-13.21	-13.28	-12.54	-26.28	-32.91
Net Interest	-0.22	-0.39	-	-	-
NPBT	-13.43	-13.67	-12.54	-26.28	-32.91
Tax expense	-	-	-	-	-
NPAT (discontinued operations)	2.07	-1.48	-	-	-
NPAT	-11.36	-15.14	-12.54	-26.28	-32.91

Balance Sheet *A$'000s*	FY23a	FY24a	FY25e	FY26e	FY27e
Cash	9.23	3.52	9.47	11.37	16.91
Receivables	7.81	6.25	5.00	8.00	7.95
Other	1.64	-	0.50	1.75	2.80
Current assets	18.68	9.77	14.97	21.12	27.66
Receivables	2.82	0.06	0.50	2.28	3.00
PPE	1.84	0.38	0.30	0.30	0.39
Intangible assets and Other	0.68	0.17	0.54	0.91	1.20
Non-current assets	5.35	0.61	1.34	3.49	4.59
Total assets	24.03	10.38	16.31	24.61	32.25
Trade and other payables	4.72	4.32	4.18	8.76	9.30
Borrowings	2.04	0.16	-	-	-
Other	1.27	0.98	-	-	-
Current liabilities	8.03	5.45	4.18	8.76	9.30
Borrowings	6.32	0.08	-	-	-
Other liability	0.12	0.17	-	-	-
Non current liabilities	6.43	0.25	-	-	-
Total Liabilities	14.47	5.70	4.18	8.76	9.30
Net Assets	9.56	4.68	12.14	15.85	22.95
Contributed Equity	389.70	399.32	419.32	449.32	489.32
Retained earnings	-404.45	-419.60	-432.14	-458.42	-491.33
Reserves/Other	24.31	24.95	24.95	24.95	24.95
Total equity	9.56	4.68	12.14	15.85	22.95

Statement of Cashflows *A$'000s*	FY23a	FY24a	FY25e	FY26e	FY27e
Net profit for period	-11.36	-15.14	-12.54	-26.28	-32.91
Depreciation & Amortisation	1.85	0.23	-	-	-
Changes in working capital	-	-0.61	0.52	-0.33	0.46
Other	-	0.26	-	-	-
Operating cash flow	-9.51	-15.26	-12.03	-26.61	-32.45
Payments for PPE	-0.14	-0.01	-	-	-
Acquisition payments	-	-	-	-	-
Proceeds from asset sale	0.01	1.49	-	-	-
Investing cash flow	-0.13	1.49	-	-	-
Equity Raised	10.00	10.00	20.00	30.00	40.00
Transaction costs	-0.74	-0.68	-1.00	-1.50	-2.00
Lease liability payments	-2.25	-2.11	-0.24	-	-
Borrowings	-	-	-	-	-
Other	-0.03	-0.02	-	-	-
Financing cash flow	6.98	7.20	18.76	28.50	38.00
Free cash flow	**-9.64**	**-13.78**	**-12.03**	**-26.61**	**-32.45**
Cash flows	**-2.66**	**-6.58**	**6.73**	**1.89**	**5.55**
Effects of exchange rate	0.72	0.09	-	-	-
Cash year end	9.23	2.74	9.47	11.37	16.91

Investment Fundamentals	FY23a	FY24a	FY25e	FY26e	FY27e
Liquidity					
Quick Ratio	1.2	1.1	1.3	1.1	1.2
Solvency					
Debt to Equity	0.9	0.1	0.0	0.0	0.0
Debt to Assets	0.3	0.0	0.0	0.0	0.0
LT Debt to Assets	0.3	0.0	0.0	0.0	0.0
Profitability					
Net Margin	n/a	n/a	n/a	n/a	n/a
ROA	-47%	-88%	-94%	-128%	-116%
ROE	-119%	-213%	-149%	-188%	-170%
Valuation					
P/E	n/a	n/a	n/a	n/a	n/a
EV/EBITDA	n/a	n/a	n/a	n/a	n/a
P/B	3.69	6.12	10.70	9.71	7.87

Key Risks

Clinical Development Risk

Syntara's lead candidate, SNT-5505, and other pipeline assets remain in various stages of clinical development. The success of these programs depends on positive outcomes in ongoing and future clinical trials. Key risks include efficacy and safety concerns, as future trials may not confirm the promising early-stage data for SNT-5505. Unexpected safety issues or suboptimal efficacy could delay or terminate development. The process of obtaining regulatory approval is uncertain and subject to stringent requirements from agencies such as the FDA and TGA. Even with positive trial results, regulatory hurdles could delay market entry. Clinical trials are expensive and time-consuming. Delays in patient recruitment, trial design issues, or unforeseen adverse events could hinder the progress of Syntara's pipeline.

Competitive Landscape

Syntara operates in a highly competitive environment, particularly in the myelofibrosis and broader haematology/oncology spaces. The presence of existing market leaders, such as JAK inhibitors like ruxolitinib and newer entrants like fedratinib, pacritinib, and momelotinib, poses a challenge. SNT-5505 will need to demonstrate superior efficacy or safety to capture market share. Other companies are actively developing novel therapies for myelofibrosis and related conditions. Competitive advancements could diminish Syntara's commercial opportunity if superior treatments emerge before SNT-5505 gains approval. Even with regulatory approval, gaining traction in the market will require substantial commercial efforts, including physician education, reimbursement approvals, and effective sales strategies.

Funding Risk

As a clinical-stage biotech company, Syntara relies on external funding to advance its pipeline. The company will require additional funding to complete late-stage trials and support commercialization efforts. While the recent $15M capital raise extends runway to mid-2026, further funding will likely be needed. Additional capital raises could lead to shareholder dilution if new equity is issued at a discount. The biotech sector is highly sensitive to market sentiment. Negative clinical trial results, macroeconomic conditions, or shifts in investor appetite for speculative stocks could impact Syntara's ability to raise capital on favorable terms.

Commercialisation & Market Access

Even if SNT-5505 and other assets successfully complete clinical development, challenges remain in bringing them to market. Securing reimbursement agreements with government and private payers is crucial for commercial success. Pricing pressures or unfavorable reimbursement terms could limit market adoption. Syntara may seek strategic partnerships for commercialization. The ability to secure favorable deals depends on clinical data strength and market conditions. Scaling up production to meet commercial demand introduces operational risks, including supply chain disruptions and quality control challenges.

IP & Legal

Syntara's ability to protect its proprietary technology and assets is essential for maintaining competitive advantage. While Syntara holds patents covering its lead assets, challenges from competitors, generic entrants, or patent litigation could erode exclusivity. Changes in regulatory policies, patent disputes, or unexpected legal hurdles could impact the commercialization pathway

Macroeconomic & Sector-Specific Risks

External factors could also influence Syntara's trajectory, including economic conditions, market downturns, inflationary pressures, and interest rate fluctuations that could impact investor sentiment and funding availability. The biotechnology sector is subject to rapid shifts in investor confidence, driven by clinical trial outcomes, regulatory changes, and broader healthcare trends. Global supply chain disruptions, international trade tensions, or regulatory changes in key markets could introduce additional uncertainties.

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