SYNTARA™

Investor Deck

Gary Phillips, CEO

July 27th 2026

FORWARD LOOKING STATEMENT

This document contains forward-looking statements, including statements concerning Syntara's future financial position, plans, and the potential of its products and product candidates, which are based on information and assumptions available to Syntara as of the date of this document. Actual results, performance or achievements could be significantly different from those expressed in, or implied by, these forward-looking statements. All statements, other than statements of historical facts, are forward-looking statements.

These forward-looking statements are not guarantees or predictions of future results, levels of performance, and involve known and unknown risks, uncertainties and other factors, many of which are beyond our control, and which may cause actual results to differ materially from those expressed in the statements contained in this document. For example, despite our efforts there is no certainty that we will be successful in developing or partnering any of the products in our pipeline on commercially acceptable terms, in a timely fashion or at all. Except as required by law we undertake no obligation to update these forward-looking statements as a result of new information, future events or otherwise.

INVESTMENT HIGHLIGHTS

Australian-founded **clinical stage drug developer**

Backed **by specialist healthcare investors** – 44% institutional

Multiple shots on goal from additional Phase 2, Phase 1 and preclinical assets

Funded into FY28 with **near term data to drive value** over the next 12-18 months. (June 26; $13.5m)

Focus on first-in-class and best-in-class drugs backed by **in house long-life patent portfolio**

Experienced team with **proven track record** in licensing deals – $100m raised

Three Phase 2 studies in **blood cancer indications** with addressable market value >$4.5 bn

$11.5m in non-dilutive grant funding awarded in last 3 years

POSITIVE 52 WEEK TOP LINE DATA FROM PHASE 2 BLOOD CANCER TRIAL

Safety and tolerability of amsulostat, together with the increasing size and durability of clinical benefit seen beyond 24 weeks, compares favourably with other drugs in clinical development

SHAREHOLDERS & CASH

Financial Information (ASX: SNT)	
Share price – 23 July 2026	$0.024
Market cap	A$47m
Cash balance (30 Jun 2026)	A$13.5m
Enterprise value	A$33.5m

Institutional Ownership	30 Jun 26
D&A Income Limited	18%
Platinum Investment Management Limited	12%
Total Institutional Ownership	**> 44%**

Research Coverage	
Canaccord Genuity	Euroz Hartleys
Bell Potter	Evolution Capital

Share Price & Volume - YTD

* 11 August 2025 recorded volume was 119,820,710 following announcement of FDA guidance for amsulostat

THREE ASSETS CREATE MULTIPLE PATHS TO VALUE

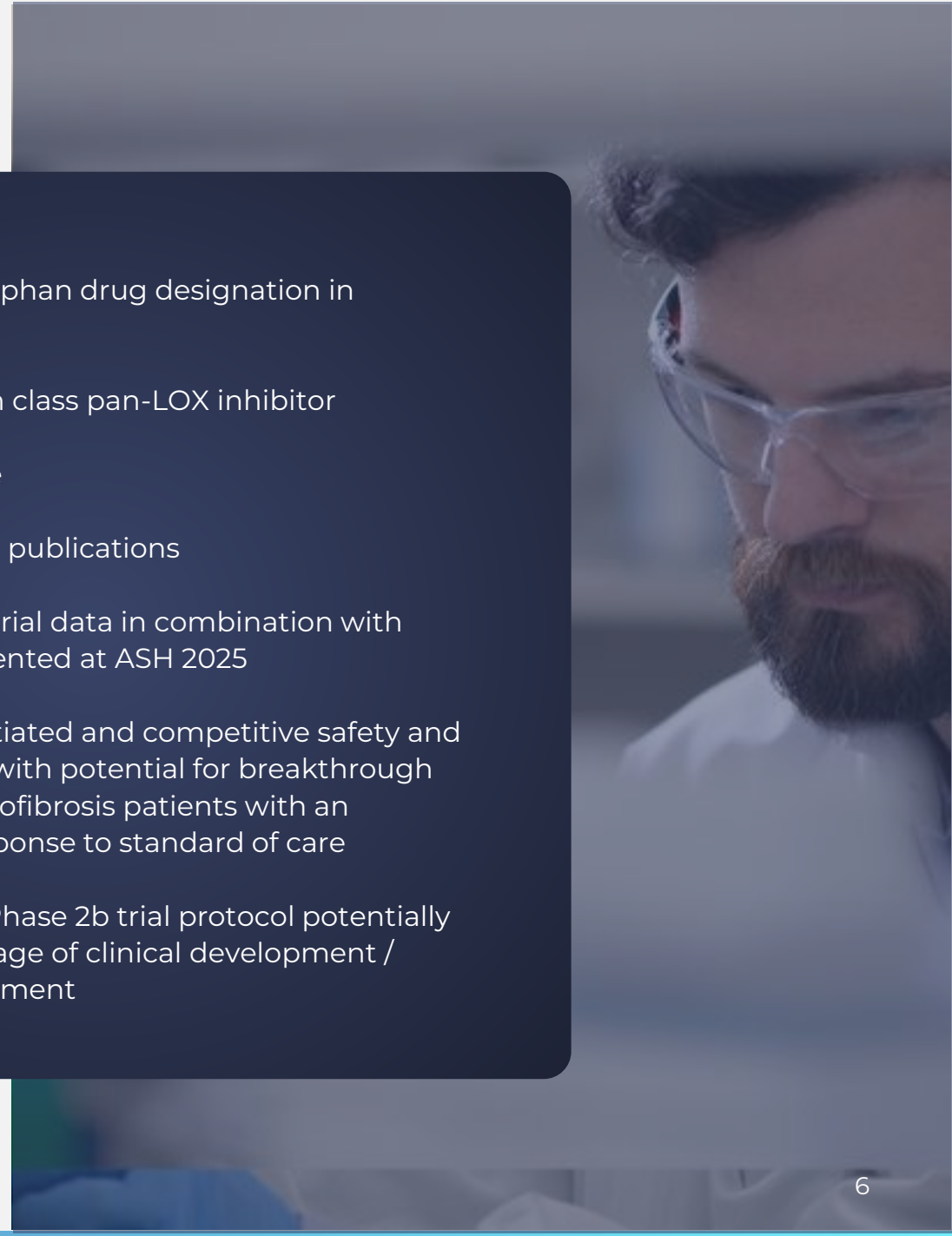
A diversified clinical-stage pipeline across blood cancers, fibrosis and neurodegenerative disease

ASSET	INDICATION	TOTAL ADDRESSABLE MARKET	DEVELOPMENT STATUS
 **amsulostat**	**Haematological malignancies**	Myelofibrosis: ~US$2.8b p.a. Myelodysplastic syndrome: ~US$3.2b p.a.	• Phase 2a clinical proof of concept in MF - complete • FDA-aligned planned Phase 2b pathway – Q4 26 start • AZALOX and MESSAGE MDS studies – preliminary data Q4 26
 **SNT-9465**	**Fibrosis and skin scarring**	Global scar-treatment market projected to reach US$50–55b by 2030.	• Hypertrophic-scar Phase 1b program – recruiting • Top-line safety and efficacy data targeted in H2 2026.
 **SNT-4728**	**Parkinson's disease and iRBD**	Global Parkinson's disease market expected to reach US$7.6b by 2030.	• Phase 2 iRBD/Parkinson's program – preliminary data reported • Final study results in Q3 2026.

AMSULOSTAT:

LEAD ASSET AND A KEY DRIVER OF VALUE

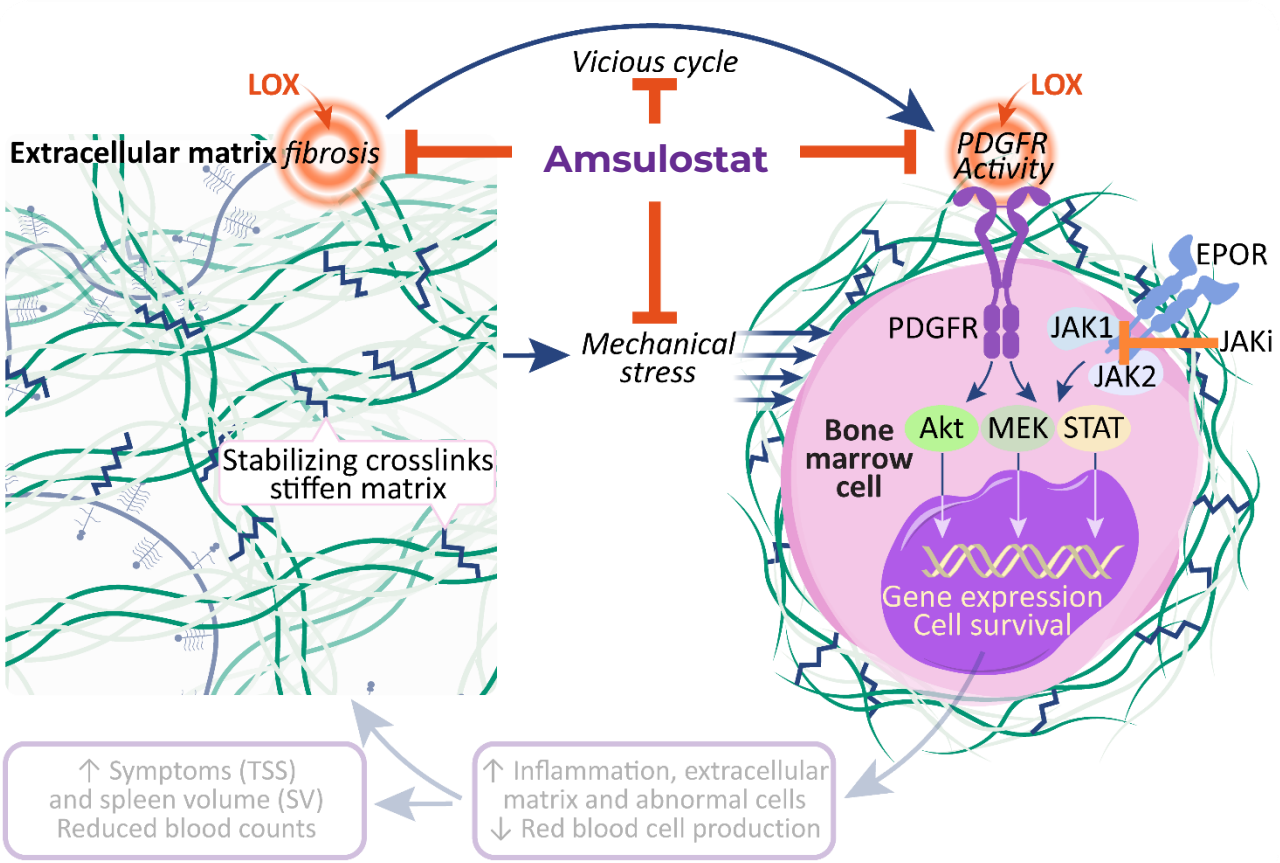
- FDA IND and orphan drug designation in myelofibrosis
- First and best in class pan-LOX inhibitor
- Long patent life
- Multiple Nature publications
- Final Phase 2a trial data in combination with ruxolitinib presented at ASH 2025
- Clearly differentiated and competitive safety and efficacy profile with potential for breakthrough therapy in myelofibrosis patients with an inadequate response to standard of care
- FDA review of Phase 2b trial protocol potentially triggers next stage of clinical development / partner engagement



LYSYL OXIDASES IN MYELOFIBROSIS

Amsulostat designed to improve the bone marrow microenvironment

Amsulostat inhibits lysyl oxidase (LOX) activity and has a multi-faceted mode of action



- ✓Inhibits cross-link formation
- ✓Reduces mechanical stress
- ✓Inhibits signalling of a growth factor that has the potential to impact JAK inhibitor efficacy

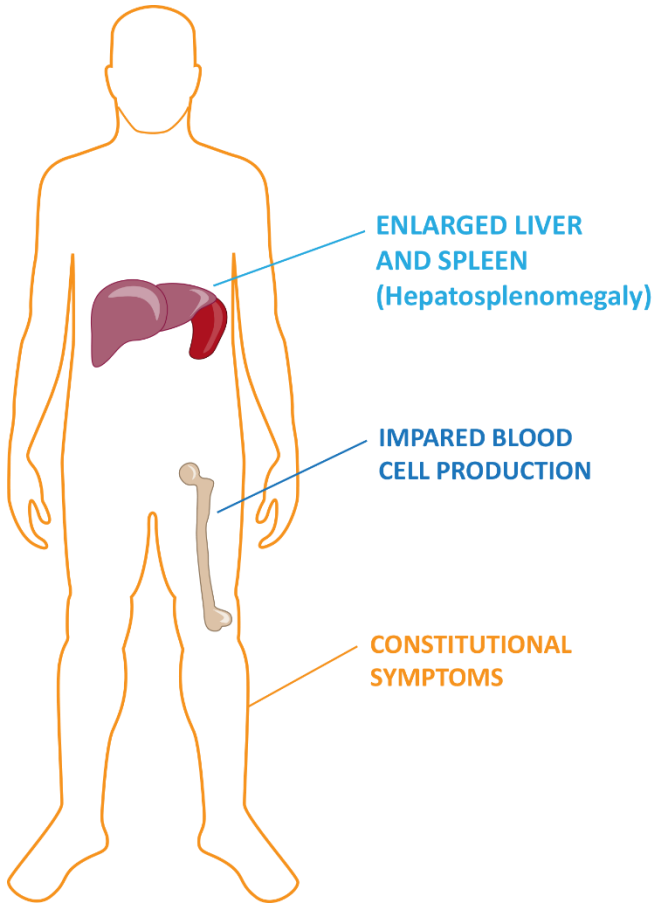
MYELOFIBROSIS

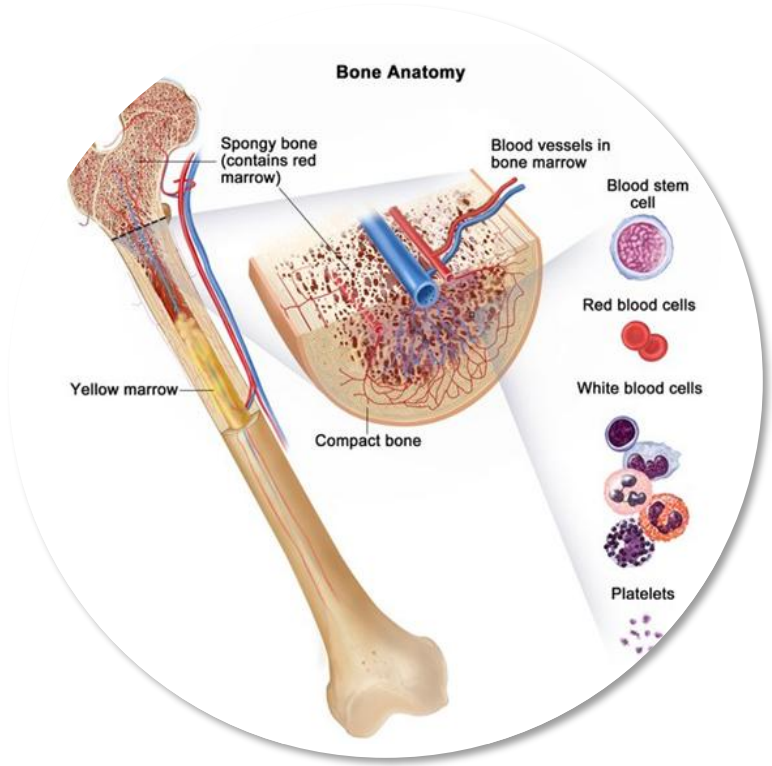
KEY FACTS

- Orphan disease affects ~55k people in 7MM (USA, Japan, 5EU); USA ~ 20,000 patients
- Age of onset typically from age 50; 5 years median survival
- 11% transformation to leukemia
- Reduced red blood cells can cause extreme tiredness (fatigue) or shortness of breath
- Reduced white blood cells can lead to an increased number of infections
- Reduced platelets can promote bleeding and/or bruising
- Enlarged spleen due to insufficient healthy blood cell production from the bone marrow causing abdominal pain
- Other common symptoms include fever, night sweats, and bone pain

A rare type of bone marrow cancer that disrupts the body's normal production of blood cells

Myelofibrosis characterised by a build up of scar tissue (fibrosis) in bone marrow and abnormal proliferation of blood precursor cells reducing the production of blood cells



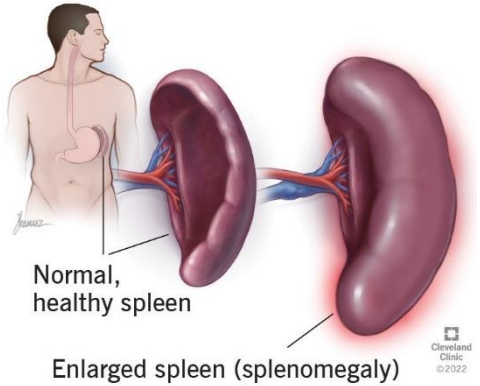


MYELOFIBROSIS

Limited treatment options currently

Current standard of care (SoC): JAK inhibitors

- Class of drugs used in the management of splenomegaly (enlarged spleen) and other constitutional symptoms



- Symptom improvement assessed using patient reported questionnaire that provides **Total Symptom Score (TSS)**
- CT or MRI scan used to measure **spleen volume reduction (SVR)**

JAK inhibitors have significant limitations

- Offer limited survival benefits and are associated with significant dose-limiting tolerability issues including cytopenias and increased risk of infection
- 75% discontinuation at 5 years
- Median overall survival only 14 – 16 months after discontinuation

Amsulostat

In contrast to SoC, amsulostat intervenes at the source, inhibiting the family of lysyl oxidase enzymes that cause increased bone marrow fibrosis and growth factor activity; both of which have detrimental effects on the production of healthy blood cells

Clinical positioning:

- ✓ Distinct mode of action
- ✓ Improved tolerability
- ✓ Profile suitable for combination with SoC
- ➢ Potential for disease modification and treatment of earlier stage disease

Commercial Opportunity

- Current SoC; revenue ~US$1.9b per annum
- Recent biotech exits after Phase 3 in excess of US$1.7b

MF-101 ADD-ON TO JAK INHIBITOR RUXOLITINIB

Heterogenous population with a high disease burden

STUDY DESIGN

- **Multi-national Phase 2a open label study to evaluate safety, PK/PD, and efficacy**
- **Int-2/high risk primary MF or post-ET/PV MF**
- **Symptomatic (TSS ≥ 10) with > 12 weeks RUX treatment**
- **Amsulostat 200 mg BID + stable dose of RUX**
- **Treatment for 52 weeks**

PATIENTS TREATED

- **16 patients recruited**
  - **11 patients completed 6 months**
  - **7 patients completed 12 months**
  - **Withdrawal rate consistent with MF studies in patients with similar disease severity**
- **Average prior RUX therapy was over 3 years**
- **High disease burden despite RUX treatment; median TSS of 23**

ENDPOINTS

PRIMARY

- Safety TEAEs

SECONDARY

- Symptom score*
- Spleen Volume Response
- Platelet response
- RUX dose modifications
- PK/PD
- Changes in BMF Grade**
- IWG Response

✓Excellent safety and tolerability profile; no drug related serious adverse events

* MFSAF v4.0 (Myelofibrosis Symptom Assessment Form v4.0; 7-day recall), assessed at baseline (BL), weeks 12, 24, 38 and 52
** Bone marrow biopsy within 3 months prior to Day 1 treatment; bone marrow biopsies scheduled at baseline, weeks 12, 24 and 52
BMF: bone marrow fibrosis; BID: twice daily; IWG: international working group; PD: pharmacodynamics; PK: pharmacokinetics; post-ET: essential thrombocythaemia; post-PV: post-polycythemia vera; RUX: ruxolitinib; TEAE: treatment-emergent adverse event

SAFETY

Amsulostat well tolerated with no treatment-related SAEs

- **Predominantly low-grade adverse events:** 74% of TEAS (63/85) were Grade 2 or lower.
- **Limited treatment-related toxicity:** 76% of TEAEs (65/85) considered unrelated to treatment.
- **No treatment-related serious adverse events:** one death reported due to unrelated SAE (congestive heart failure).
- **No hematological toxicity burden:** 9 other SAEs reported (all non-hematological and unrelated to amsulostat).
- **AEs don't require special management** unlike other agents in development, no prophylaxis required for AE management.

WHY THIS MATTERS

Profile supportive of sustained use as add-on therapy of choice.

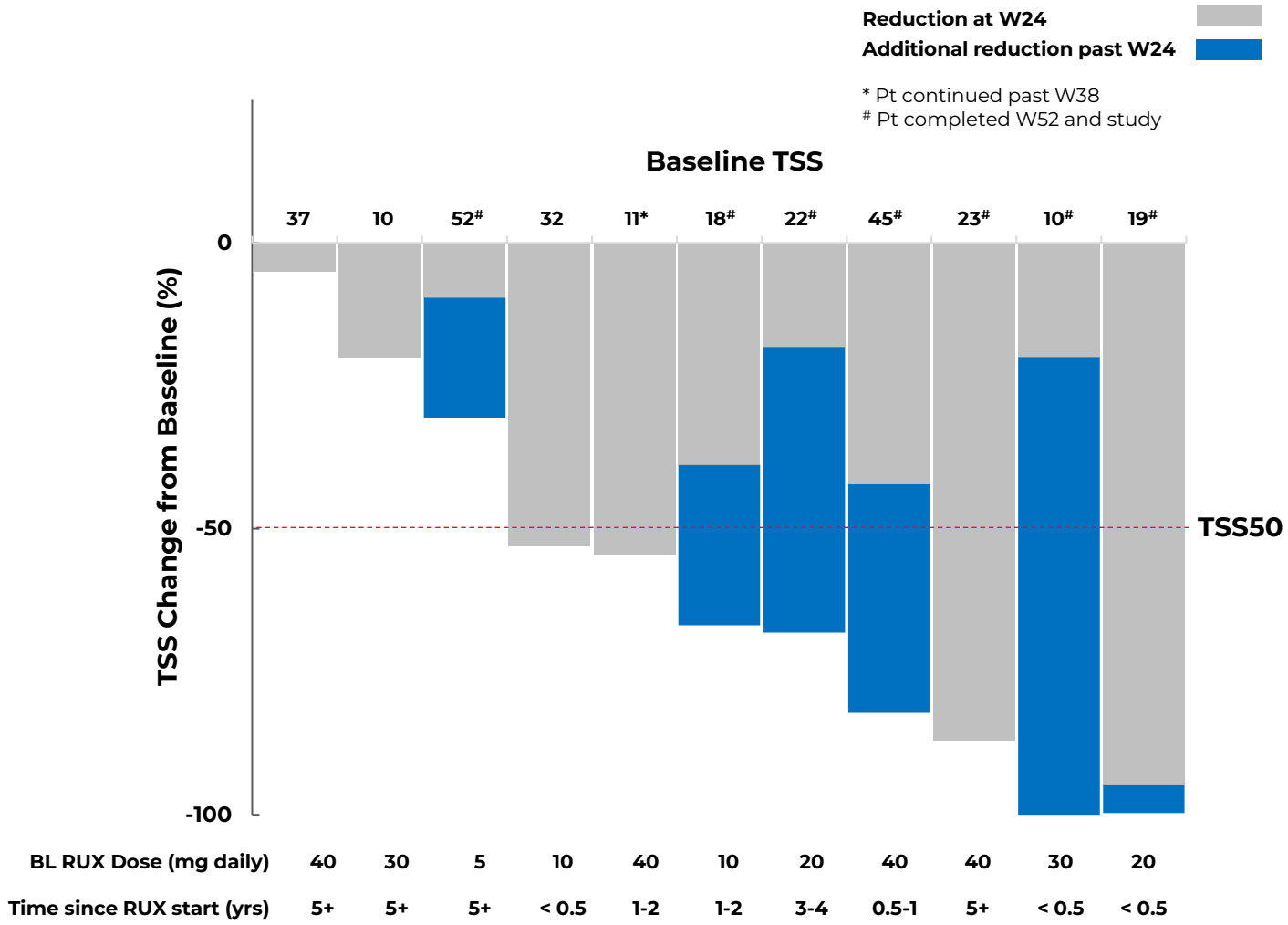
- Supports combination use with JAK inhibitors and potentially other treatment classes.
- May enable sustained treatment duration without adding substantial tolerability burden.
- Differentiates amsulostat from competitors where hematological toxicity and prophylactic management of AEs may limit dosing regimens and longer-term use.
- Simple to administer for community based hematologists

TOTAL SYMPTOM SCORE

73% (8/11) of patients achieved TSS50 at Week 24 or beyond

Symptom relief continues for patients:

- 73% (8/11) patients* achieved TSS50 at Week 24 or beyond
- Mean TSS reduction from baseline to Week 38 (n=8) was 56%
- Mean TSS reduction from baseline to Week 52 (n=7) was 68%



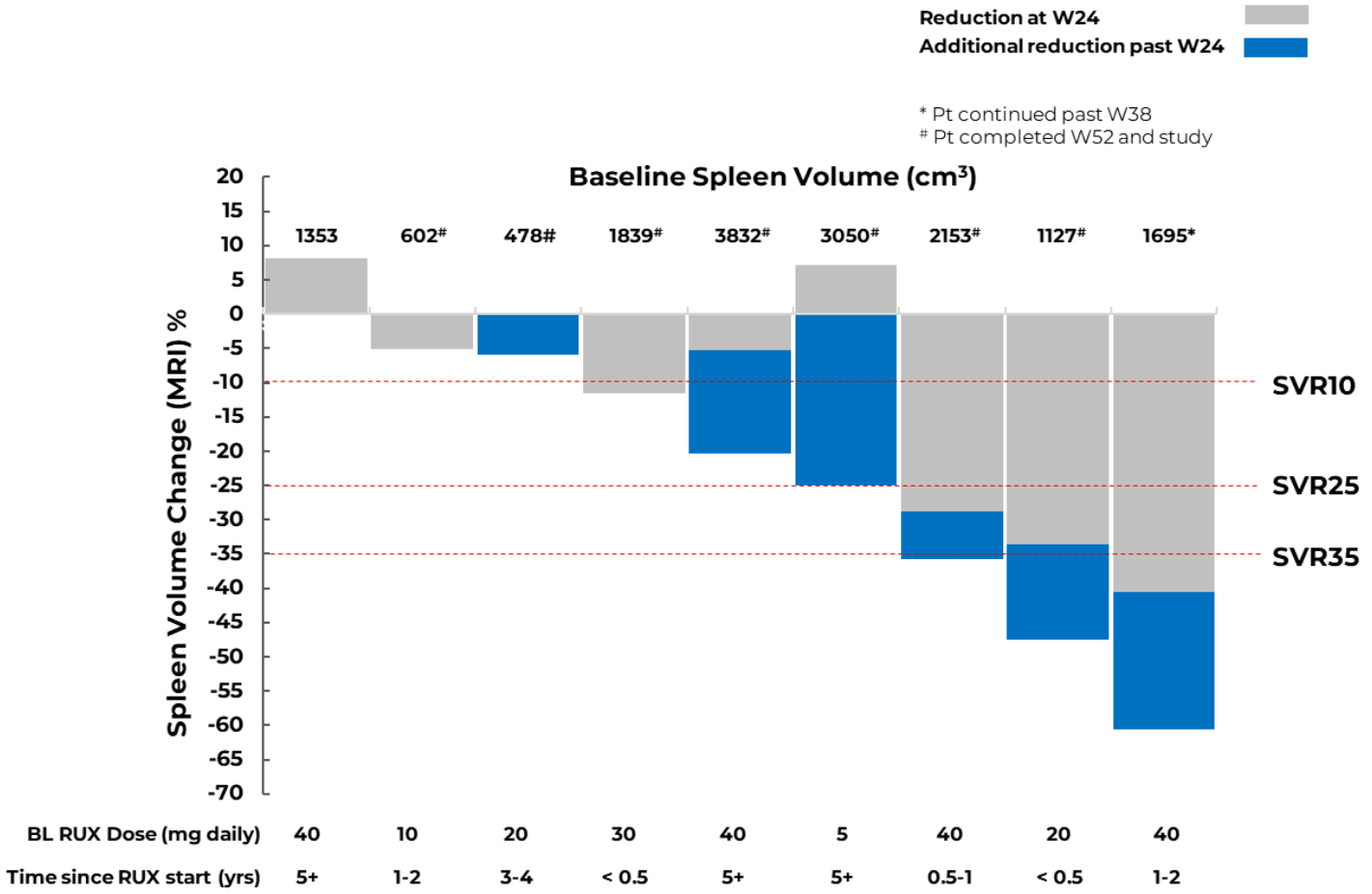
* Results for TSS50 at Week 24 or beyond are for the 11 patients reaching Week 24

SPLEEN VOLUME REDUCTION

44% (4/9) of patients achieved SVR25 at Week 24 or beyond

Improved spleen volume reduction:

- At Week 24, 7/9 (78%) evaluable patients* experienced stable or reduced spleen volume with no increases in RUX dose
- 4/9 (44%) evaluable patients achieved SVR25 at Week 24 or beyond



* Evaluable patients are those who had spleen volume ≥ 450 cm³ at baseline and with ≥ 80% RUX use

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SUMMARY AND NEXT STEPS

Top-line Phase 2a data highlights amsulostat's potential in MF

Improvements of 50% or more in total symptom score (TSS50)

were observed quickly (as early as 12 weeks) and were sustained, with 73% (8/11) of patients achieving TSS50 at Week 24 or beyond

Meaningful spleen volume reductions (SVR) were observed

at 24 weeks and maintained thereafter, with 44% (4/9) of patients achieving SVR25 at Week 24 or beyond

Of the 7 patients that completed 52 weeks of treatment

- 6 chose to continue on amsulostat through named patient supply
- 3 of these patients had a minor anaemia response
- 2 achieved a complete (100%) resolution of symptoms from baseline

Differentiated TPP

- Competitive safety and efficacy profile in suboptimal MF JAKi patients
- Potential for disease modification and use in earlier stage MF
- Positive FDA review of Phase 2b trial protocol clears next stage of clinical development

Next phase of development and study design for BRIDGE-MF guided by clinical advisory board and aligned with recent FDA feedback.

SYNTARA APPOINTED GLOBAL EXPERTS SUPPORTING AMSULOSTAT DEVELOPMENT

Strategic Advisors to Syntara Board, Haematology

- **Dr Adam Craig**
  Former CEO of CTI BioPharma Corp, responsible for taking the JAK inhibitor Vonjo (pacritinib) through clinical development, FDA approval and subsequent commercialisation. In 2022 CTI was acquired by SOBI in a deal worth US$1.7 billion.
- **Dr Kevin Lynch**
  Former Chief Medical Officer, Antengene and VP Clinical Development and Medical Affairs in Asia, Celgene; involved in early and late-stage development of haematology oncology drugs.

Myelofibrosis Clinical Advisory Board

- **Professor Claire Harrison**
  Professor of Myeloproliferative Neoplasms and Deputy Chief Medical Officer at Guy's and St Thomas' NHS Foundation Trust
- **Dr Gaby Hobbs**
  Associate Professor of Medicine, Harvard Medical School and Clinical Director, Leukemia, Massachusetts General Hospital.
- **Professor John Mascarenhas**
  Professor of Medicine at the Icahn School of Medicine at Mount Sinai, Director of the Center of Excellence for Blood Cancers and Myeloid Disorders, and a member of The Tisch Cancer Institute.
- **Professor Dr Haifa Kathrin Al-Ali**
  Medical Faculty, Martin Luther University Halle-Wittenberg

BRIDGE-MF: STUDY DESIGN

Study designed to strengthen confidence in both efficacy and disease-modifying relevance

Trial Design

Main Cohort A

- Patients with MF on ruxolitinib for min 3 months
- Current inadequate response to ruxolitinib (TSS and spleen size)
- Historical inadequate response to ruxolinitib (spleen response)

Optional Cohort B

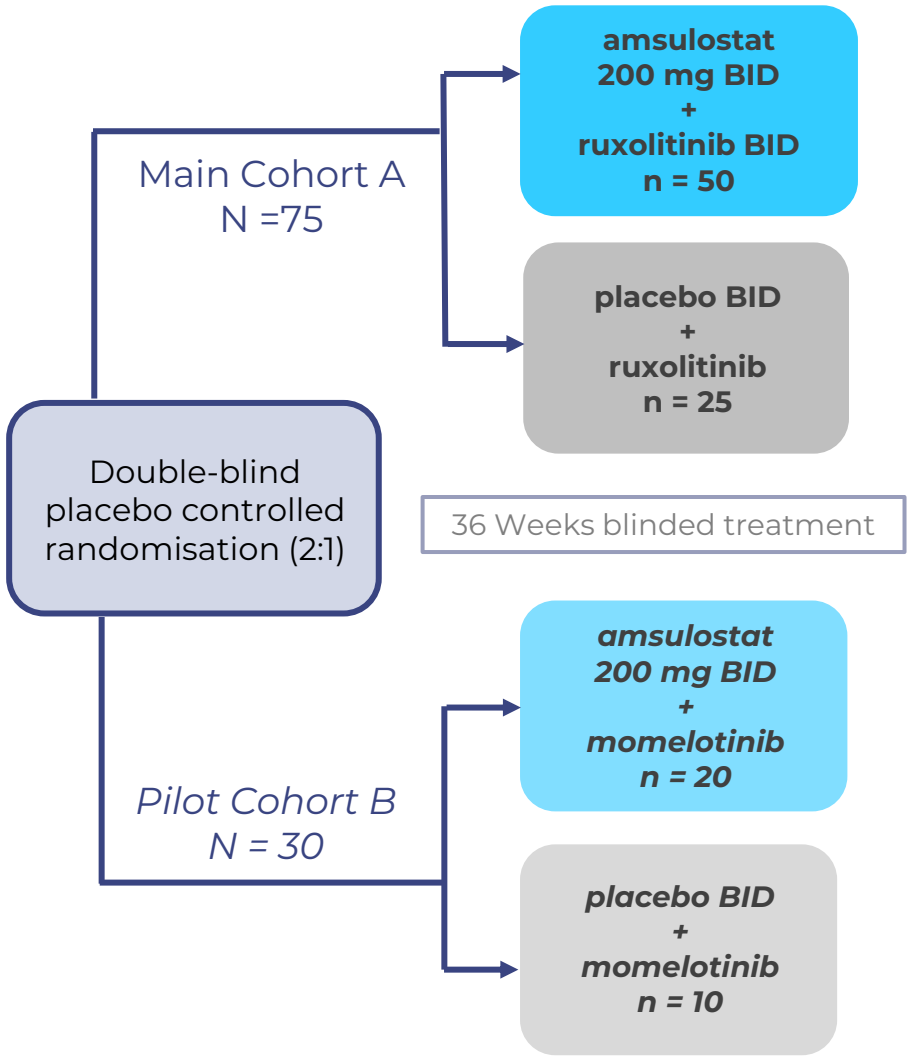
- Patients with MF with inadequate response to momelotinib

Primary Endpoint

- TSS50 at Week 36

Secondary Endpoints

- Spleen size (SVR 25 and 35)
- Other symptom assessments
- Haematological response
- BMF assessment by biopsy and MRI
- Biomarkers of inflammation and ECM remodelling
- Overall Survival
- Safety, PK/PD



Timeline of Activities

Preparation Phase

- Protocol finalisation
- CRO selection
- Trial site negotiations
- Formulation development and clinical trial supplies

Trial progression

- Commencement: Q4 2026
- Recruitment: 18 months
- Treatment duration: 9 months

Trial Budget

- US$20-25m
- GSK likely to support with free momelotinib

AMSULOSTAT: A NEW FUTURE IN MF TREATMENT OPTIONS

Aspirational lifecycle development in MF enabled by first in class pan-LOX mechanism

Ideal add-on for patients with sub-optimal response to JAKi

- FDA endorsed phase 2b protocol
- Pan-lysyl oxidase inhibition mechanism and favourable tolerability support combination use with JAKi.
- Inhibition of LOX-mediated PDGFR signalling enhances impact of JAKi on cell proliferation.
- Potential to improve symptoms and extend the useful treatment duration of JAKi through effects on the bone marrow microenvironment

Market Opportunity ~$1b

Potentially best positioned in frontline combination with JAKi

- Pan-lysyl oxidase inhibition mechanism and favourable tolerability profile makes amsulostat well suited for early combination use.
- Targeting fibrosis biology and microenvironmental signaling complements JAK inhibition.
- Early use maximises impact on symptoms, spleen response and longer-term disease biology.

Market Opportunity ~$1.8b

Opportunity to slow progression in newly diagnosed MF

- Pan-lysyl oxidase inhibition may preserve and restore the bone microenvironment to slow disease progression.
- Strong tolerability supports use earlier in treatment paradigm, before patients become symptomatic.
- Potential for disease modification and improved survival is maximised when used early.

Market Opportunity ~$2.8b

STRONG INTEREST IN MF ASSETS FROM STRATEGICS

Target / Acquiror	KARTOS THERAPEUTICS / IPSEN	Ajax / Lilly	中国生物制药有限公司 / sanofi	KEROS / Takeda	morphosys / NOVARTIS
DATE OF ANNOUNCEMENT*	**JULY-2026**	**APRIL-2026**	**MARCH-2026**	**DEC-2024**	**FEB-2024**
Drug Name	Navtemadlin	AJ1-11095	Rovadicitinib	Elritercept	Pelabresib
Lead Indication / Phase (at transaction)	Myelofibrosis (ongoing Phase 3)	Myelofibrosis (ongoing Phase 1)	Myelofibrosis (completed Phase 2)	MDS and MF (ongoing Phase 2 trials)	Myelofibrosis (successful Phase 3 studies)
Deal Type	Acquisition	Acquisition	License	License	Acquisition
Upfront / Milestones (US$)	US$450M / US$1.3B	Total deal value US$2.3B	US$135M / US$1.395B	US$200M / US$1.1B	US$2.9B
Earnout Payments / Royalty Rate (%)	Not disclosed	Not disclosed	Not disclosed	Not disclosed	Subject to regulatory approvals

Attractive commercial outcomes for drugs with Phase 2 and 3 data expected to drive interest in amsulostat Phase 2 data

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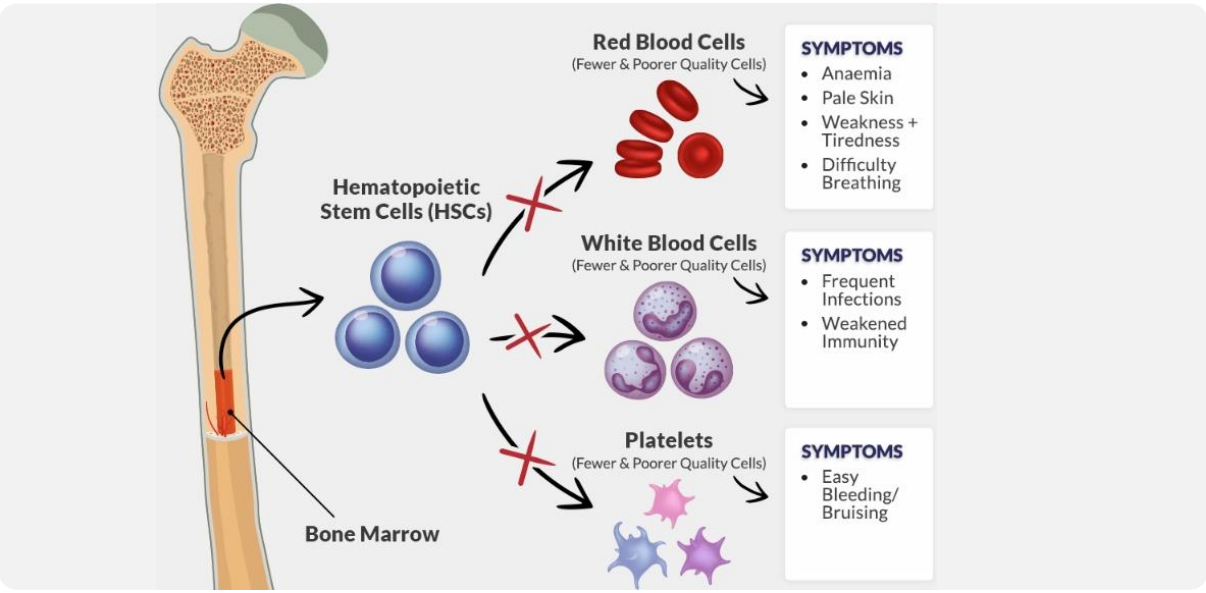
*** Prior to 2024:**
SOBI acquisition of Pacritinib from CTI for US$1.7B
GSK acquisition of Momelotinib from Sierra Oncology for US$1.9B

Myelodysplastic Syndromes (MDS)

INDICATION EXPANSION: Myelodysplastic Syndrome (MDS)

Market Opportunity ~US$3.2b p.a.

Diverse bone marrow disorders characterized by inadequate production of healthy blood cells



- 12–20k new cases are reported every year in the US (87k p.a. worldwide)
- 25–30% have high-risk MDS with average survival of ~1 year
- 1 out of 3 MDS patients progress to acute myeloid leukemia (AML)

High unmet need

- Therapy for low-risk MDS patients is aimed at improving cytopenia(s) to prevent complications
- In intermediate to high-risk MDS, SoC is hypomethylating agents (HMAs) such as azacytidine (5-AZA) or decitabine
- Only ~50% of patients respond to HMAs and most responders eventually progress; median overall survival 4–6 months after HMA failure
- Encouraging results from drugs in development in combination with 5-AZA are significantly offset by toxicity

Impressive preclinical results published in Nature

- Amsulostat on top of 5-AZA translates to significantly increased blood cell production in xenograft animal models (closest mimic of human disease)
- Response to 5-AZA (29%) is potently augmented by concomitant lysyl oxidase inhibition with amsulostat (65%)

AML: acute myeloid leukemia; 5-AZA: 5-azacytidine; HMA: hypomethylating agent; MDS: myelodysplastic syndrome; SoC: standard of care
Platzbecker U, 2021, *Leukemia*. Papadantonakis et al. 2012, *Blood;* Nowak at al. 2023, *Nature Communications*

TWO STUDIES IN MDS TO DELIVER PHASE 1b DATA - 2H 2026

Generates additional interest from strategics

AZALOX:

Phase 1b/2a open label study to evaluate safety, PK/PD and efficacy

- **High risk MDS** *or* intermediate-2 to high risk CMML
- Amsulostat in combination with 5-AZA
  - 1b: 3-12 patients treated for 24 weeks
  - 2a: 30 patients treated for 24 weeks
- Study running in Germany with support from Deutsche Krebshilfe

PRIMARY ENDPOINT

- Safety, TEAEs

SECONDARY ENDPOINTS

- Including haematological response, disease progression and survival

MESSAGE:

Phase 1b/2a open label study to evaluate safety, PK/PD and efficacy

- Transfusion-dependent **low/intermediate risk MDS**
- Amsulostat in combination with oral decitabine
  - 1b: 3-12 patients treated for 24 weeks
  - 2a: 18 patients treated for 24 weeks
- Study running in Australia with support from ALLG

PRIMARY ENDPOINT

- Safety, TEAEs

SECONDARY ENDPOINTS

- Including transfusion independence, haematological response, quality of life and survival

ALLG: Australasian Australasian Leukaemia & Lymphoma Group; CMML: chronic myelomonocytic leukaemia

SNT-4728: BRIEF HISTORY

Safe and efficacious, small molecule enzyme inhibitor that crosses the blood brain barrier

- Syntara discovered and wholly owned asset
- Drug extensively de-risked from earlier collaboration with Boehringer Ingelheim
- Once daily dosing achieves consistent, dose-dependent inhibition of two enzyme targets; SSAO and MAO-B.

SNT-4728 FOR NEUROINFLAMMATION

A novel approach

- Strong preclinical and clinical data support the role of SSAO and MAO-B as **drivers of neuroinflammation**
- **Dual SSAO & MAO-B** inhibition provides an opportunity for anti-inflammatory and neuroprotective treatments through the combination of proven and safe mechanisms.

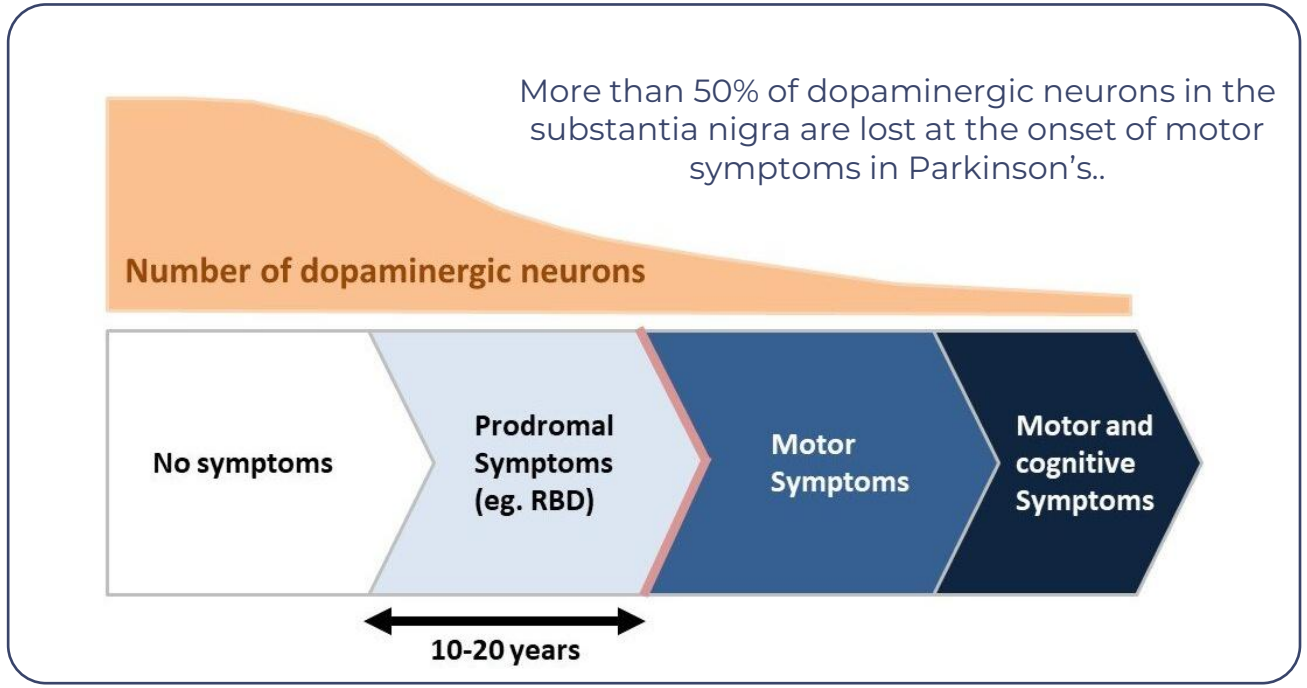
iRBD AND PARKINSON'S

KEY FACTS

- Patients with isolated REM sleep behaviour disorder (iRBD) act out their dreams, often violently.
- IRBD can represent a prodromal stage of Parkinson's
- Prodromal symptoms precede the onset of motor cognitive dysfunction.
- > 70 % of iRBD patients go on to develop Parkinson's and the related α-synuclein deposition disorders, dementia with Lewy bodies (DLB) and multiple system atrophy (MSA)
- Annual conversion rate from iRBD to overt neurodegenerative condition is 6.25%[1]
- > 8% of 70 – 89 year olds have iRBD.
- Unmet medical need as there is currently no approved treatment; current standard of care is melatonin.

Using a sleep disorder to target Parkinson's.

Early intervention to demonstrate neuroprotection – rather than late treatment when most neurons have already been lost (for example in PD) – is where disease-modifying, slowing therapies are likely to have their greatest impact.



[1] *Risk and predictors of dementia and parkinsonism in idiopathic REM sleep behaviour disorder: a multicentre study*; doi: https://doi.org/10.1093/brain/awz030

SNT-4728: CLINICAL TRIAL

Phase 2 multi-centre, placebo-controlled study assessing effect of SNT-4728 on brain inflammation in patients with iRBD

40 patients (3:1 drug to placebo randomisation), drug treatment duration 12 weeks, 15 mg oral, once daily

Key inclusion criteria: definitive iRBD, mild (sub-clinical) parkinsonism and either/both hyposmia or reduced colour discrimination.

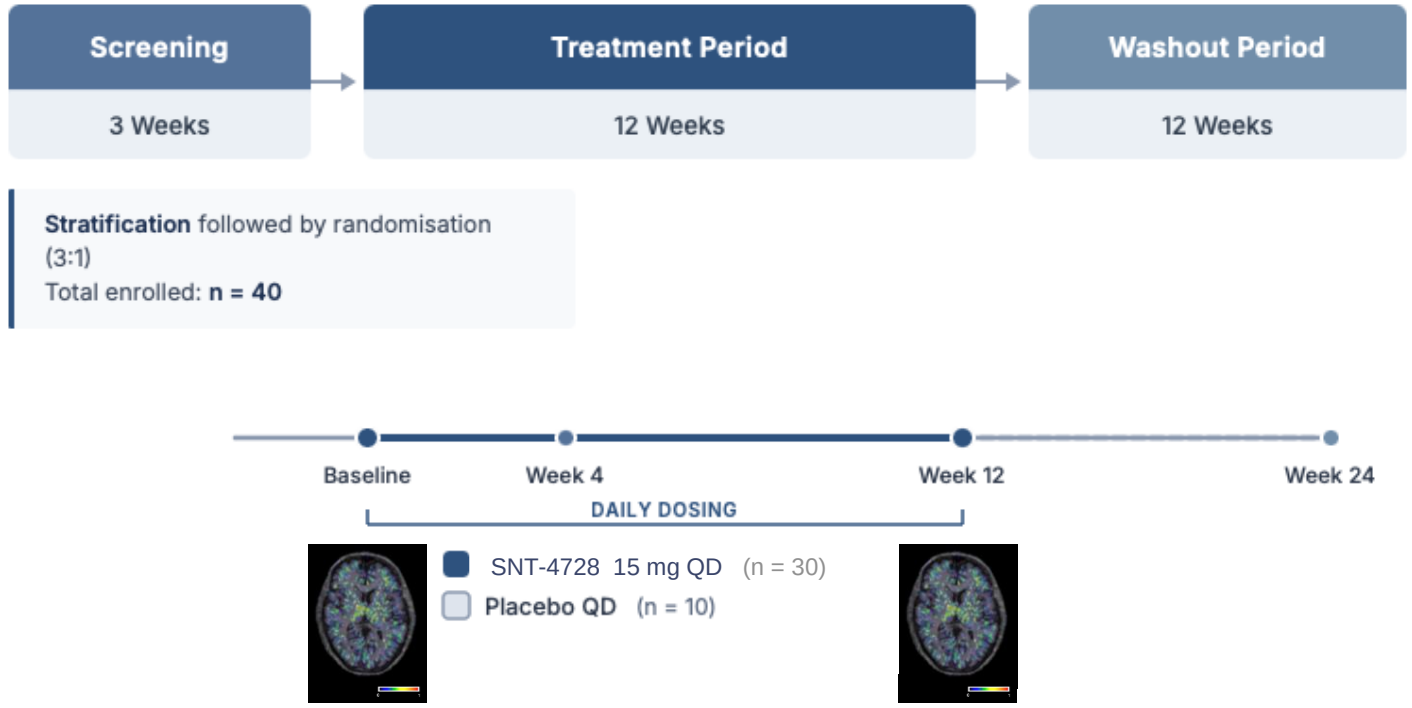
Follow-up period of 12 weeks.

ENDPOINTS

- Safety TEAEs up to Week 24
- Change in neuroinflammation measured by PET ligand binding in brain regions of interest at Week 12
- Digital and biological markers up to Week 24

Parkinson's UK

Study funded by Parkinson's UK Virtual Biotech and is being run at sites in Sydney, Australia and Oxford, UK



PET: positron emission tomography; TEAE: treatment emergent adverse event; QD: once daily.

BASELINE CHARACTERISTICS

Study successfully recruited an enriched iRBD population at high risk for phenoconversion to Parkinson's and related diseases

- Median age of study participants was 68 yrs and 93% were male.
- 90% of participants were hyposmic/anosmic (reduced or loss of sense of smell).
- 46% of participants had low colour discrimination.
- 95% had misfolded alpha-synuclein detected in cerebrospinal fluid.

EXPOSURE AND SAFETY

In keeping with the excellent safety profile observed in prior Phase 2 studies, SNT-4728 was safe and well tolerated

- All 41 patients completed randomised treatment period.
- Baseline and safety data not unblinded as study ongoing.
- Across the treatment arms, 125 TEAEs in 27 patients – all Grade 1 or 2; none serious.
- Only 14 TEAEs considered related in 5 patients.
- Most common TEAEs were headache (19.5% of patients), nasopharyngitis (9.8%), muscle spasms (7.3%), constipation (7.3%), dizziness (7.3%).

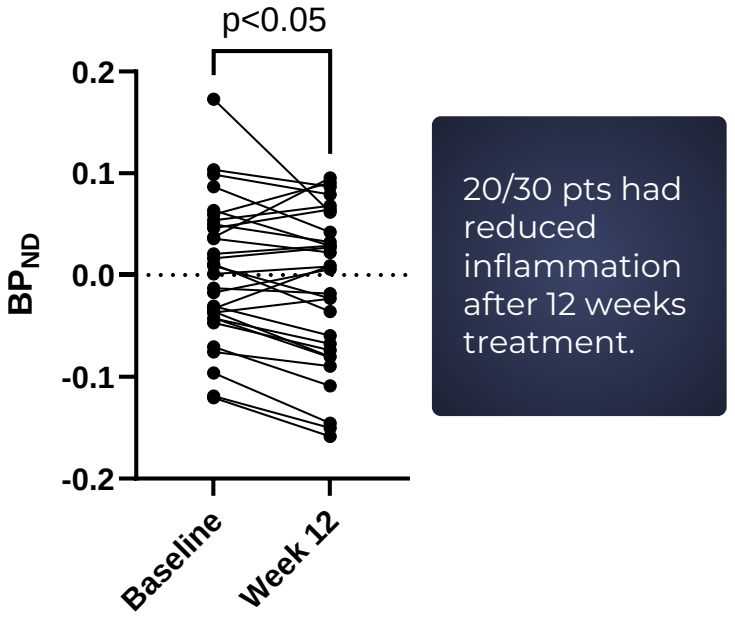
TEAE: treatment-emergent adverse events.

REDUCTION IN INFLAMMATION

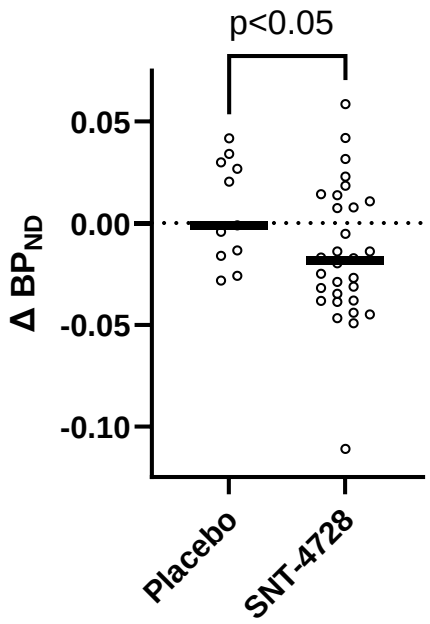
Statistically significant reduction in brain inflammation observed unilaterally in the putamen (p-value 0.0145)

- The putamen is a key region underlying the hallmark motor symptoms of Parkinson's disease.
- 20 out of 30 pts on active treatment (SNT-4728) recorded a reduction in inflammation from baseline.
- No statistically significant change in inflammation was detected in other predefined regions of interest.
- No significant reduction in brain inflammation in any region for placebo group.

Putamen	Binding Potential (BP_{ND})		Mean change	P-value (Paired t-test)
	Mean at baseline	Mean at 12 weeks		
Right	0.002918	-0.012073	**-0.016 (0.033)**	**0.0145**
Left	-0.001209	0.001692	0.003 (0.029)	0.6219



20/30 pts had reduced inflammation after 12 weeks treatment.



At week 12, inflammation significantly reduced compared to placebo.

BP_{ND} is a dimensionless number expressing the distribution volume ratio of specifically bound radioligand to that of non-displaceable radioligand.

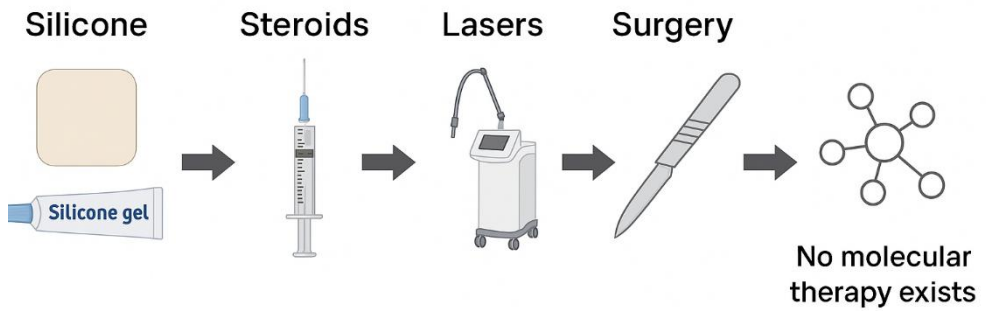
SKIN SCARRING

Leverage expertise in LOX inhibition

- Skin Scarring is a Global Disease
- 100 million new scars annually post surgery in the developed world alone
- Surgical, traumatic, burn, acne, cosmetic, obstetric etc.
- A lifelong condition with functional + psychological impact
- No FDA-approved therapies for scar treatment & remodeling
- The global scar treatment market projected to reach ~$50 billion by 2030

Standard of Care: Fragmented, Ineffective and Outdated

- Silicone sheeting (none to very limited effect)
- Steroid injections (temporary; high relapse)
- Laser therapy (expensive; outcome operator dependent)
- Surgery (expensive, recurrence common, especially in keloids)



Opportunity: A Downstream, Mechanically Targeted Therapy

- Only LOX inhibition addresses matrix stiffness
- LOX crosslinking is a shared downstream driver of fibrosis across organs; a biological bottleneck with almost no therapeutic competition
- Pan-LOX inhibitors have already shown ECM remodeling in humans

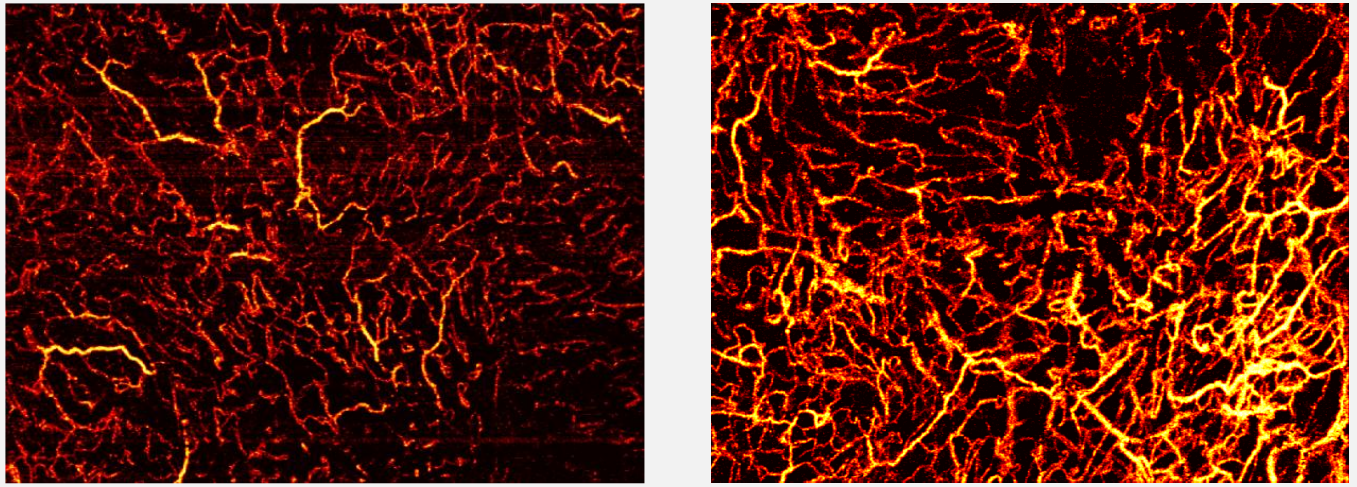
CLINICAL PROOF OF CONCEPT ALREADY ACHIEVED

Placebo controlled study with first generation topical pan LOX inhibitor significantly de risks program

*"We now understand the biology well enough to **target stiffness directly** rather than just calming inflammation around it. LOX activity sits at the **mechanical root** of scar persistence, and its involvement is consistent across scar types. When we **inhibit LOX,** **we see meaningful structural reversal** in human studies"*

Professor Ardeshir Bayat
Director Medical Research Council of South Africa Wound Healing Unit, University of Cape Town.

SOLARIA2 Findings*



Images at day one (L) and day 90 (R) show a significant increase in blood vessel density following SNT-6302 treatment, that is similar to normal uninjured skin.

- Patients in the active arm had a mean reduction in collagen of 30% compared to placebo after three months treatment. ($p<0.01$).
- Advanced non-invasive imaging technology reveals pan-LOX inhibition leads to extracellular matrix remodelling and significant improvement in scar vascularisation
- SNT-6302 treated scars become structurally and biologically closer to normal uninjured skin
- No changes observed for placebo-treated patients

SYNTARA

Skin scarring webinar with Syntara and Professor Bayat available here: https://youtu.be/7ijx3wnpN6A?si=4OT7k95RfOhlJHYZ
Morellini N. et al 2025 *Sci. Transl. Med. A randomized, double-blind, placebo-controlled phase 1 trial of the topical pan–lysyl oxidase inhibitor PXS-6302 in mature scars.*

INNOVATIVE TRIAL DESIGN TO EVALUATE COMMERCIAL VIABILITY

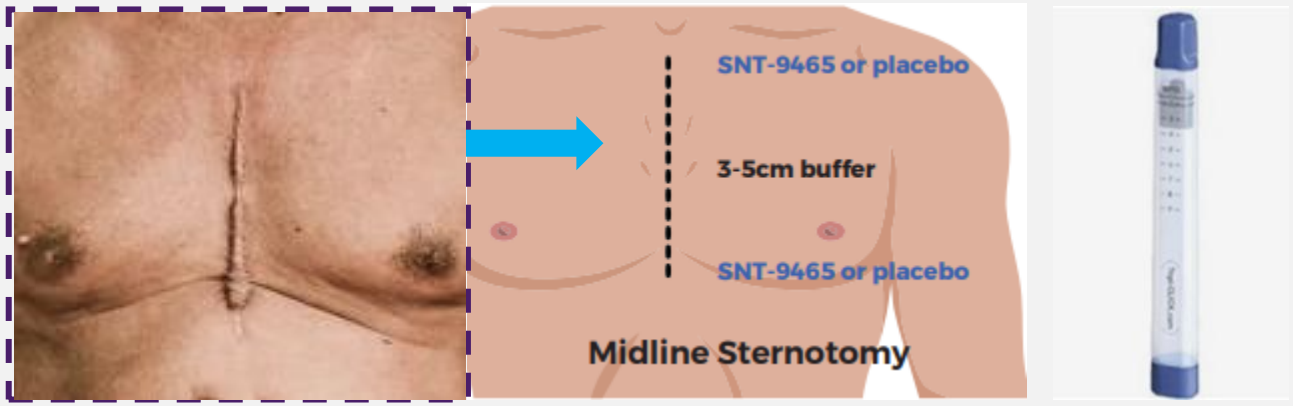
Placebo controlled scar remodeling study with multiple efficacy readouts in H2 2026

Healthy Volunteer Phase 1a Single Ascending Dose

✓ Dose-dependent target engagement confirmed

Hypertrophic scars Phase 1b Multiple Daily Dose

Randomized, Double-blinded,
Placebo-controlled Split-scar Study in Adult Participants with Hypertrophic Sternotomy Scars (N=20)



Trial design optimised for inclusion criteria and endpoints

PRIMARY ENDPOINT

- Safety TEAEs

SECONDARY ENDPOINTS

- Pharmacokinetics / Pharmacodynamics
- Scar rating (multiple blinded raters)
- 3D imaging (scar volume)
- Optical Coherence Tomography (OCT)
- Elastography
- Patient and Observer Scar Assessment Scale (POSAS)

THE YEAR AHEAD - *POISED TO DELIVER NEAR TERM VALUE*

TARGET	DRUG	INDICATION	PARTNERS	PHASE 1	PHASE 2	ANTICIPATED NEWS FLOW	
				HEALTHY PARTICIPANTS	PATIENTS	H1 2026	H2 2026
Pan-LOX	**Amsulostat (SNT-5505)**	Myelofibrosis		→	→	FDA approved development plan and partner engagement	Trial Progression
		High Risk MDS AZALOX trial	Deutsche Krebshilfe	→	→		Interim safety and efficacy data Phase 2 initiation
		Low / Int Risk MDS MESSAGE trial	ALLG	→	→		Interim safety and efficacy data
		Pancreatic cancer FALCON trial	Garvan Institute of Medical Research	→	→		Investigator Study TBD
Topical Pan-LOX	**SNT-9465**	Hypertrophic scarring		→	→	Recruit hypertrophic scar Phase 1b trial	Top Line safety and efficacy data
	SNT-6302	Keloid scarring	The University of Western Australia, Rona Wood Foundation	→	→		Interim safety and efficacy data
Dual SSAO & MAO-B	**SNT-4728**	IRBD / Parkinson's Disease	In partnership with Parkinson's UK	→	→	Phase 2 Top Line data	Phase 2 full results

SYNTARA

MDS: Myelodysplastic Syndrome; Int: Intermediate; IRBD: Isolated REM sleep Behaviour Disorder

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