

SELLAS Life Sciences Group

Corporate Presentation

November 2026

NASDAQ: SLS

SELLAS LIFE SCIENCES GROUP

Forward Looking Statements

This presentation contains forward-looking statements. Such forward-looking statements can be identified by the use of the words "expect," "believe," "will," "anticipate," "estimate," "plan," "project" and other words of similar import. The forward-looking statements in this presentation include, without limitation, statements related to the clinical development programs of galinpepimut-S (GPS) and SLS009 (tambiciclib), clinical data of GPS and SLS009, the pre-clinical development of SLS009, plans for further clinical development of SLS009, the potential for GPS and SLS009 as drug development candidates and anticipated milestone dates. These forward-looking statements are based on current plans, objectives, estimates, expectations and intentions, and inherently involve significant risks and uncertainties. Actual results and the timing of events could differ materially from those anticipated in such forward-looking statements as a result of these risks and uncertainties, which include, without limitation, risks and uncertainties associated with oncology product development and clinical success thereof, the uncertainty of regulatory approval, and other risks and uncertainties affecting SELLAS and its development programs. These risks and uncertainties are described more fully under the caption "Risk Factors" in SELLAS' Annual Report on Form 10-K filed on March 19, 2026 and in its other filings with the Securities and Exchange Commission (the "SEC"). Other risks and uncertainties of which SELLAS is not currently aware may also affect SELLAS' forward-looking statements. The forward-looking statements herein are made only as of the date hereof. SELLAS undertakes no obligation to update or supplement any forward-looking statements to reflect actual results, new information, future events, changes in its expectations or other circumstances that exist after the date as of which the forward-looking statements were made.

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SELLAS Mission: Extending Patient Lives with Novel Therapies

Two Clinical Stage Assets | **Each Potentially First & Best in Class** | **AML Lead Indication for Both**

Galinpepimut-S (GPS): Innovative Immunotherapy Engineered to Target Wilms Tumor 1 (WT1) Antigen

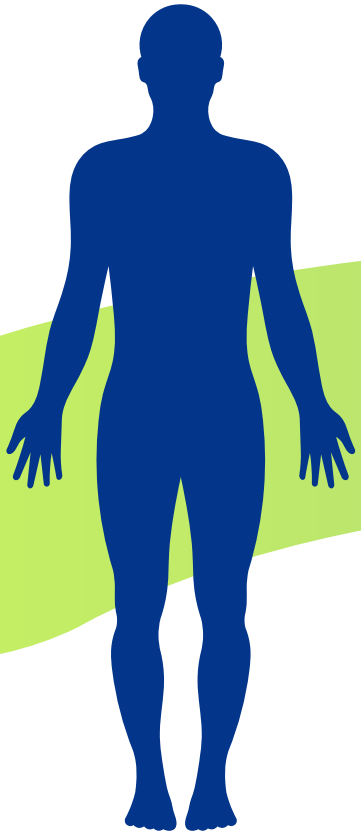
- In November 2026, SELLAS announced that pivotal Phase 3 REGAL trial met its primary endpoint with GPS demonstrating a clinically meaningful and statistically significant reduction in risk of death by 41%, with median Overall Survival of 24.0 months versus 14.7 months for best available treatment, and a Hazard Ratio of 0.59 (p=0.035) in AML patients who have achieved a second complete remission (CR2)
- Company plans to meet with the U.S. Food and Drug Administration (FDA) while advancing preparations for an expedited Biologics License Application (BLA) for GPS
- Phase 1/2 combination trials completed with positive data in combination with anti-PD1 drugs in advanced ovarian cancer and metastatic pleural mesothelioma (MPM)
- Orphan Drug Designation (ODD) from the FDA & EMA, Fast Track, and Rare Pediatric Disease Designation (RPDD) from the FDA

SLS009 (tambiciclib): Novel Highly Selective CDK9 Inhibitor

- Phase 2 trial in combination with aza/ven in relapsed and/or refractory (r/r) AML: Nov. 2026 announced data from its completed Phase 2 study of SLS009 (tambiciclib) for the treatment of relapsed or refractory acute myeloid leukemia (r/r AML) will be presented at the 68th American Society of Hematology (ASH) Annual Meeting. All primary endpoints met:
  - Patients with r/r monocytic AML achieved a 54% ORR with a median overall survival (mOS) of 5.8 months and patients with TP53-mutated AML achieved a 57% ORR and a mOS of 8.8 months, comparing favorably to historical outcomes in these adverse-risk settings of ~2.5 months
  - Successfully overcomes venetoclax resistance: SLS009 targets two primary drivers of venetoclax resistance: TP53 mutations and MCL-1 reliance. The combination demonstrates robust anti-leukemic activity in difficult-to-treat genetic and phenotypic populations following venetoclax failure.
  - Study showed that SLS009 (tambiciclib) combination was well-tolerated with no meaningful added toxicity
- In September 2026, presented preclinical data at AACR 2026 Conference on Pancreatic Cancer: SLS009 demonstrated substantially greater single-agent activity than daraxonrasib in the daraxonrasib-resistant model, inducing 17.9% apoptosis versus 2.8% with daraxonrasib and 14.5% necrosis versus 2.7%.
- 40 patients now enrolled in Phase 2 trial of SLS009 in combination with aza/ven in frontline AML; preliminary topline data expected in Q4 2026
- ODD from FDA & EMA, Fast Track & RPDD from FDA; more than ~180 patients treated with no safety issues

AML Treatment Overview and Addressable Market

GPS and SLS009 Have Potential for Broad AML Applicability



PATIENT JOURNEY

AML Diagnosis
~77K patients/year[1]
~97% of AML patients express WT1

1st Line Therapy
80% patients[2]
Chemotherapy, Targeted Therapy

SLS009 in AML
Ph2 initiated 1Q26

Complete Remission 1 (CR1)
50-55% patients[2]
Maintenance Therapy:
Chemotherapy, Targeted Therapy
Stem Cell Transplant

Potential GPS Label Expansion: post CR1 and post transplant

1st Relapse; 2nd Line Therapy
30-35% patients[2]
Chemotherapy, Targeted Therapy

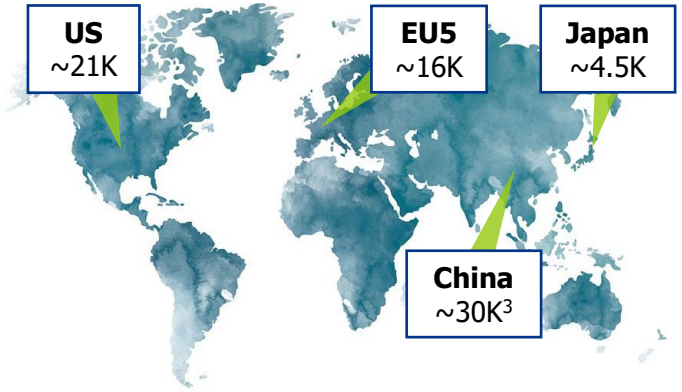
SLS009 in r/r AML
Ph2a completed 2Q25

Complete Remission 2 (CR2)
12-15% patients[2]
Maintenance Therapy:
Chemotherapy, Targeted Therapy

Primary Endpoint Met: GPS in Ph3 REGAL Trial

CR2 population expected to grow over foreseeable future with increased therapeutic clinical benefit derived for prior line patients

Annual AML Incidence (Adults)[1]



Notes: 1. Global Data forecasts for 2020 2. Patient segmentation incidence adapted from Kurosowa. S et al. (2010), Haematologica. 95(11): 1857 – 1864; 3. James Moore, Patientworthy.com, July 25, 2019

GPS

Innovative WT1 Targeting Immunotherapy

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GPS: Innovative Technology with Broad Immuno-Oncology Applicability

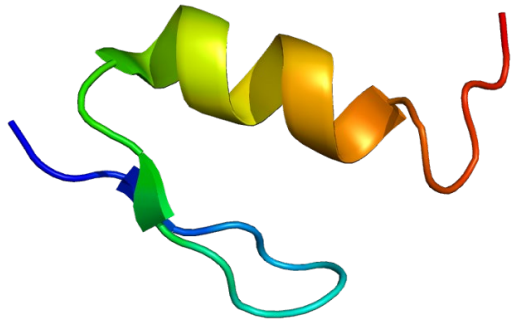
GPS Drug Candidate

- **Highly engineered** immunotherapy designed to **increase immunogenicity** and **break tolerance**
- Heteroclitic multivalent mixture of engineered and artificially mutated **peptides targeting 25 select WT1 epitopes**
- **CD4 and/or CD8** immune responses across the majority of HLA types
- Exclusive license from Memorial Sloan Kettering Cancer Center
- Composition of matter patent to at least 2033
- Potential to be used as **monotherapy** or **in combination** with other immuno-oncology (I/O) agents in multiple solid and hematological tumor types

GPS Targets Tumors Expressing WT1 Protein

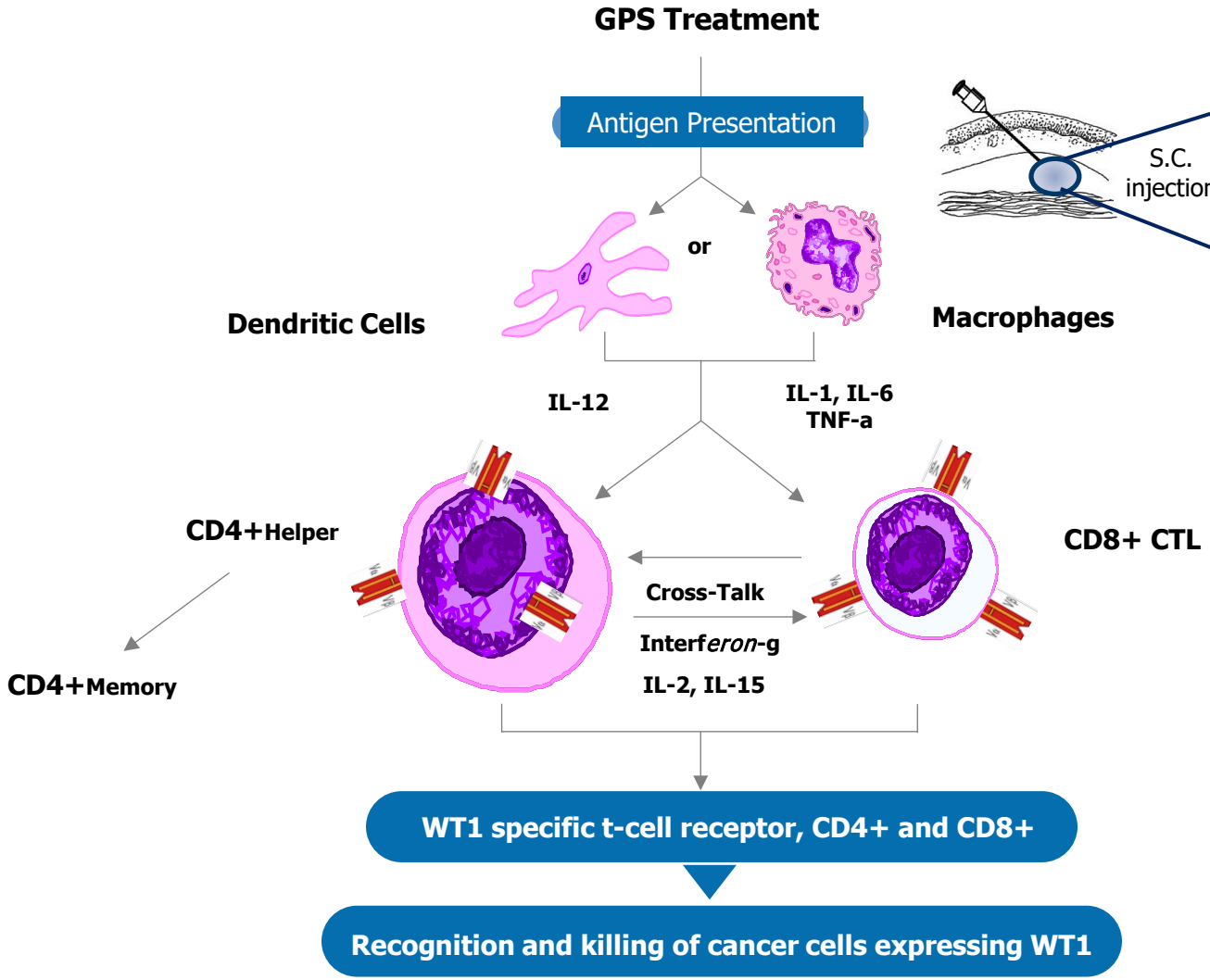
Wilms Tumor 1 Protein

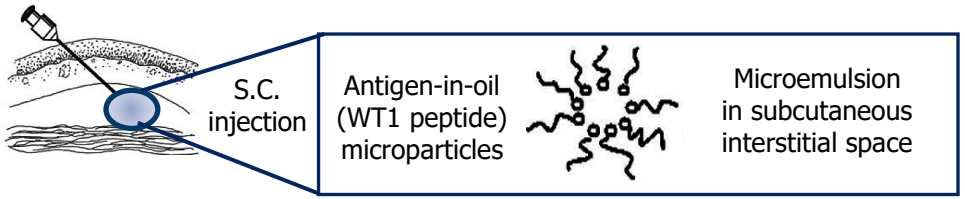
- WT1 is one of the most widely expressed cancer proteins in multiple malignancies
- Intracellular oncofetal antigen, WT1 not expressed in adult tissues, which **lowers potential off-target toxicity**
- Potential to treat **20+ hematological** and **solid tumor** cancers
- Densely and almost universally expressed in AML: **~97% express WT1**



[1]'The Prioritization of Cancer Antigens: A National Cancer Institute Pilot Project for the Acceleration of Translational Research' Cheever et al; Clin Cancer Res., 2009.

GPS Designed to Promote Immune Response and Sustain Remission







Mixture of 2 native and
2 synthetic WT1 peptides

Produces both CD4+ (memory, helper) and CD8+ (tumor killing) WT1-specific activated cells

- Multi-epitope, broad cross-reactivity to entire WT1 protein
- Applicable to majority of human HLA types (~90%)

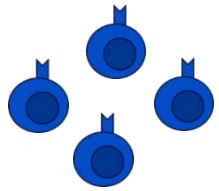
Sources: Gomez-Nunez M,et al. LeukRes.2006;30:1293-8; Pinilla-IbarzJ, et al. Leukemia. 2006;20:2025-33; SchijnsVE, et al. Curr. Protoc. Immunol. 2014;106:2.18.1-7.

GPS-Treated Lymphocytes Reactive to WT1 Protein

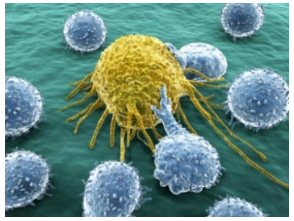
Epitope Spreading:

A Key Element for Cancer Immunotherapy

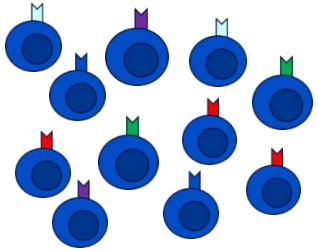
GPS-specific T-cells recognize and kill tumor cell



Tumor cell death releases new antigens into tumor microenvironment



New antigens create T-cells specific for broader antigen set that host was not initially immunized against



Evidence of epitope spreading in GPS treated patients[1]:

Lymphocyte reactivity to WT1 protein epitopes for which patient was not specifically immunized[2]

- **CD8+ T cells specific to GPS peptides:** up to ~91% show antigen-specific T-cell (CD8 and/or CD4) immune response against at least one of the constituent WT1 peptides
- **CD8+ and CD4+ T-cells reactive to additional WT1 epitopes, that were not from GPS:** High frequency (83.3%) of T-cell responses to WT1-derived antigens (~240 epitopes) against which patients were not specifically inoculated

Results suggest:

- Active cancer cell killing
- Release and presentation of WT1 peptide fragments
- Processing of additional WT1 epitopes processed by immune synapse
- Production of a broader, *de novo* expanded, repertoire of CD8+ clones specific to epitopes not included in GPS

[1]Koehne G, et al *EBMT* 2018. [2]Patients treated with GPS tested against overlapping peptide epitopes of the entire WT1 protein (~113 pentadecapeptide fragments; ~230 epitopes; "WT1 – All pool")

GPS

Clinical Development in AML

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REGAL Phase 3 Registration Enabling Clinical Trial in CR2 AML

Target Population and Inclusion Criteria

AML in CR2/ CRp2[1] or later CR patients ≥18 yrs
N=125 - 140

Ineligible for/unable to undergo allogeneic stem cell transplant (Allo-SCT)

~95 centers WW

Stratification axes:

- CR2 vs CRp2 status
- Cytogenetics risk category at initial diagnosis (poor vs all other)
- Duration of CR1 ≥ 12months versus < 12 months
- Minimal Residual Disease (MRD) Status

Treatment: Open Label, Randomized Multi-Center Phase 3 Trial

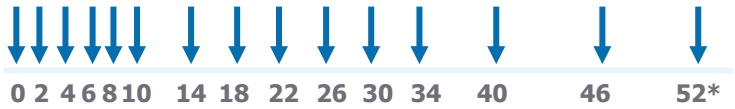
GPS

Montanide (500 µL/dose)

GM-CSF (70 µg/dose; d-2 & d0)

Schedule of Administration (weeks)

R: 1:1



*Every 6 weeks thereafter until disease relapse

Best Available Therapy (BAT): Clinician's choice of observation; or HMAs and/or venetoclax; and/or low-dose Ara-C

Endpoints; Interim/Final Analysis

Primary Endpoint: Overall Survival

Secondary Endpoints:

- Leukemia free survival (LFS)
- OS and LFS landmark rates
- MRD
- MRD by multigene array
- WT1-specific T-cell (CD8/CD4) immune response (exploratory)

Interim and Final Analysis: Statistical Analysis Plan (SAP) provides for planned interim safety and futility analysis after first 60 events (deaths) and final analysis after 80 events (deaths)

Notes: 1.CRp2: CR2 with incomplete platelet recovery, i.e., platelet count of ≥60 x 10^9/L (as defined for this study)

Positive REGAL Phase 3 Topline Data Announced on November 17, 2026

Primary Endpoint Met

Galinpepimut-S (GPS) demonstrated a statistically significant and clinically meaningful improvement in overall survival (OS) versus investigator's choice of best available treatment (BAT).

REGAL STUDY DESIGN

Phase 3, randomized 1:1, global trial

N = 126

GPS vs investigator's choice of BAT

Indication

- AML in CR2 or later CR/CRp; transplant-ineligible
- Significant unmet need for treatment approaches
- Fast Track and ODD designations

TOPLINE EFFICACY

41%

reduction in risk of death

Median OS (months)

GPS		BAT
24.0	vs	**14.7**

Hazard ratio 0.59

p = 0.035

Primary endpoint analysis of intent-to-treat population

TOPLINE SAFETY

- Generally well tolerated
- Consistent with prior clinical experience; no new safety signals identified
- Treatment-related adverse events (AEs) predominantly mild-to-moderate (Grade 1/2)
- Most common: local injection-site reactions

Primary endpoint analysis conducted following the prespecified occurrence of 80 events (deaths). NCT04229979: GPS maintenance in transplant-ineligible AML in CR2+/CRp2+; investigator-selected BAT included observation, HMA, venetoclax and/or low-dose cytarabine.

Overall Survival in GPS Treated AML CR1 Patients Far Exceeded Historical Comparators

Phase 2 Open-label Clinical Trial in AML CR1 Patients

Overall Survival (OS)
GPS vs **Standard of Care (SOC)**
including allogeneic stem cell transplant

All Age Patients

67.6 months vs **17.5 - 25 months**

>60 Years Patients

35.3 months vs **9.5 - 16.8 months**

Key Points:

- Two clinical trials in CR1 AML completed: Phase 1 (N=10), Phase 2 in CR1 (N=22)
- Primary endpoint of 3-year OS > 34% was met with 3-year OS in 47% of patients. Historical OS for AML Cr1 patients at 3 years is ~25%[1]
- Phase 2 survival with GPS in elderly (>60 yr) AML patients in CR1 far exceeded that seen with historical comparators, even allogeneic stem cell transplant
- 88% of patients dosed with GPS had an antigen-specific immune response to any of the 4 GPS peptides of either CD4+ and/or CD8+; ~50% had CD4+ response at both early and late time points
- CD4+ responses seen across all HLA-Class II subtypes tested
- No significant safety issues observed; no myelosuppression observed

Source: CR1 Phase 2 study - Maslak et al., Blood Adv. 2018. Notes: 1. In cohorts of patients who have undergone induction and post-CR consolidation chemotherapy (across all ages, assuming an Allo-SCT rate of ~25%).

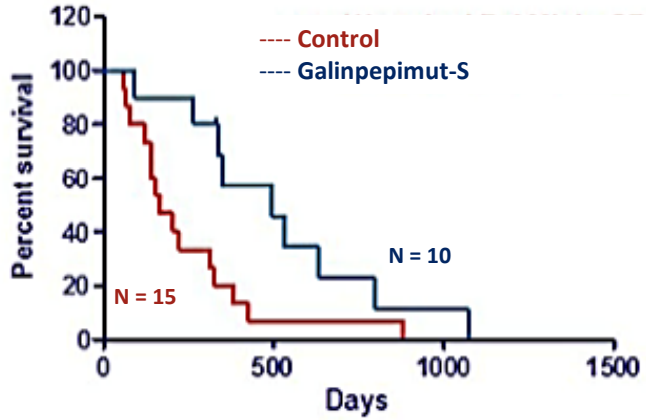
Survival Benefit Seen in AML Patients in CR2

Phase 2 Open-label Clinical Trial in Contemporaneously Treated AML CR2 Patients at Moffitt Cancer Center (MCC)[1,2]

Median Overall Survival (All Age Patients):**GPS** vs **SOC**

21.0 months vs **5.4 months** (p<0.02)

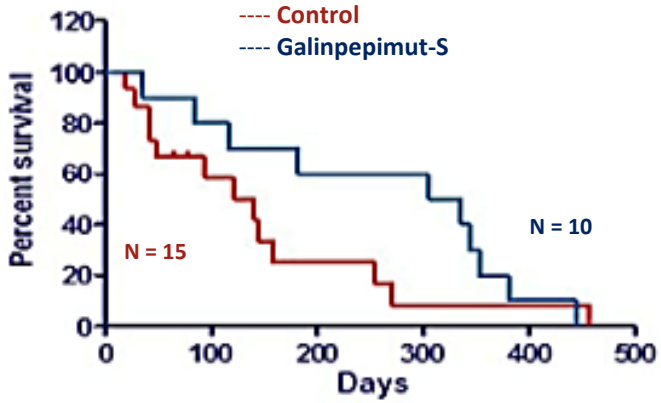
OVERALL SURVIVAL



Relapse Free Survival (All Age Patients):**GPS** vs **SOC**

10.5 months vs **4.3 months**

RELAPSE-FREE SURVIVAL



- Median age→ in GPS arm = 74 years; in control arm = 73 years; % Male patients → 80% in GPS arm; 66.7% in control arm
- All 25 patients met hematologic (morphological) criteria for their second complete remission (CR2).
- All 25 patients had measurable WT1 transcript by PCR at the time of enrollment, and, therefore, were all MRD(+), i.e., had evidence of minimal residual disease
- No patients in either cohort had undergone an allogeneic stem cell transplant, either in CR1 or in CR2
- High-risk cytogenetics at initial diagnosis (%) → 40% in GPS arm; 33.3% in control arm; intermediate-risk cytogenetics at initial diagnosis (%) → 50% in GPS arm; 53.3% in control arm
- No significant safety issues observed; no myelosuppression observed

Notes: 1. Brayer et al., Am J Hematol. 2015; 2. SELLAS, Data on file, 2020.

GPS

Combination with Checkpoint Inhibitors

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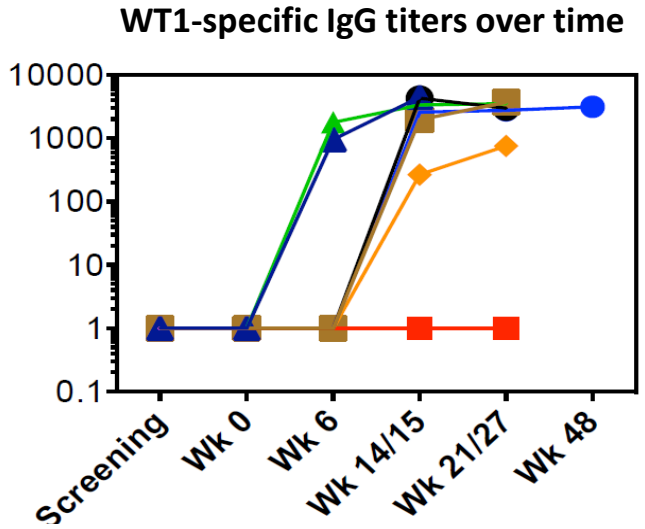
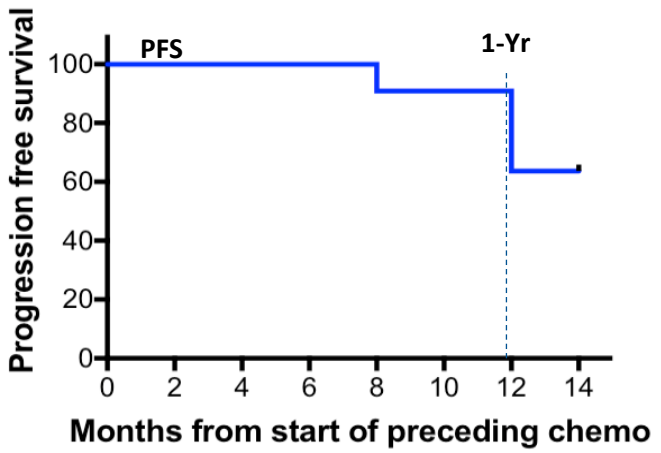
GPS Activity Demonstrated in Combo with Checkpoint Inhibitors in WT1 Expressing Solid Tumors

Open-label Phase 1 Clinical Trial of GPS + Nivolumab in MRD Negative, WT1+ Ovarian Cancer Patients After 1st or 2nd Salvage Chemotherapy

Key Points:

- Primary Endpoint met: GPS + nivolumab was well tolerated in study (n=11). Most frequent treatment related adverse events (TRAEs): injection site reaction, joint pain, and fatigue. None above Grade 2
- Secondary Endpoint met: WT1-specific IgG (against all 4 GPS peptides) observed in 86% of patients (wks 6 – 27). $CD4^+$ and $CD8^+$ T cell responses also observed (wks 6 – 15)
- Exploratory Endpoint: Landmark 1-year PFS rate = 70% in pts who received >1 dose of GPS + nivolumab (n=10). Historical PFS rates[2-4] do not exceed 50% in this setting[1]
- Exploratory Endpoint: Landmark 2-year PFS rate = at least 30% in pts who received >1 dose of GPS + nivolumab (n=10)[5]. Historical PFS rates[2-4] range between 3-10% in this setting[5]





Notes: 1. O'Cearbhaill RE. ASCO 2018; Abstr. 5553; 2. Parma, Lancet, 2003; 3. Harrison, Gynecol Oncol. 2007; 4. Sabbatini, Gynecol Oncol. 2010; 5. SELLAS, data on file (updated PFS analysis; Nov. 2019).

GPS Combination With Checkpoint Inhibitor: R/R Ovarian Cancer

Open-label Phase 1/2 Clinical Trial GPS + Pembrolizumab

Confirmatory final data presented November 2023:

- Disease Control Rate (DCR) (overall response rate + rate of stable disease) of 50.1% for combination at a median follow-up of 14.4 months vs. 37.2% in pembrolizumab KEYNOTE-028 study in a similar patient population
- Median PFS for the combination of 12 weeks compared to 8.4 weeks in pembrolizumab KEYNOTE-028 study in a similar population
- Median OS of 18.4 months compared to 13.8 months in pembrolizumab KEYNOTE-028 study in similar patient population
- Safety profile similar to pembrolizumab alone
- Survival and DCR benefits observed in patients with any level of detectable PD-L1 expression
- Correlation observed between PFS and OS and WT-1 specific immune response after GPS vaccination

GPS in active disease setting

- GPS + pembro combination appears to be effective in active disease state to halt or slow down progression
- Has been primarily investigated in previous trials as maintenance therapy after patients reach MRD- complete remission

Large unmet need in this patient population

- Very difficult to treat patients with active disease who underwent intensive chemotherapies with no apparent clinical benefit

GPS in Combination With Checkpoint Inhibitor: MPM

Open-label Phase 1 IST of GPS + nivolumab in relapsed/refractory MPM post 1L SOC Therapy (Pemetrexed) at MSKCC: Primary endpoints of safety and efficacy met

STUDY POPULATION (n=10)

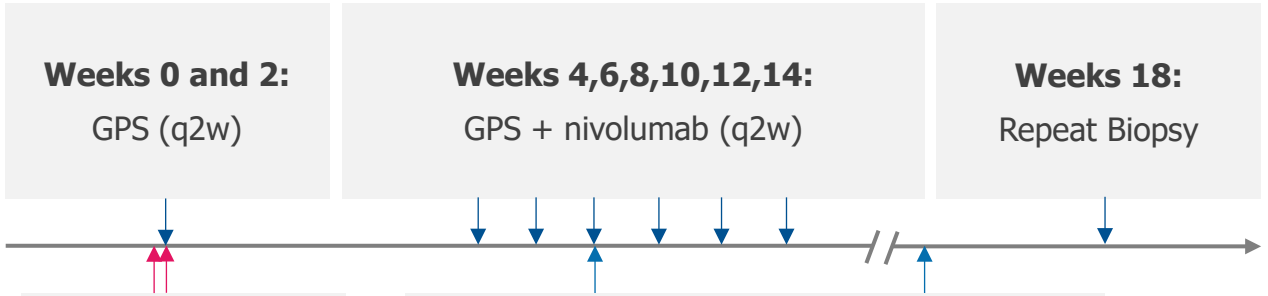
Metastatic or recurrent MPM

KPS ≥ 70%

WT1+ on immunohistochemistry (IHC)

1 prior course of pemetrexed-based chemo

Baseline biopsy ≤ 60 days before treatment starts



CONTINUATION

Additional GPS + nivolumab x 6 cycles if no disease progression

- **Median OS of 70.3 weeks in 9 patients who received combination**; median OS in R/R patients with standard of care is approximately 28 weeks; median progression-free survival was 11.9 weeks for all 10 patients; safety of combination similar to that seen with nivolumab alone
- Median OS among patients who did not have an immune response (IR) to GPS was 9.0 months; **the median OS for patients who had an IR to GPS was 27.8 months, more than three times longer median OS (208.3% increase).**
- Among the nine evaluable patients, CD4+ IR (44.4%), CD8+ IR (33.3%) to GPS. Both CD4+ and CD8+ IR (33.3%).
- Among patients who had a full IR (both CD4+ and CD8+) to GPS, 66.7% achieved an objective response, while among the patients who did not have an IR to GPS, 14.3% achieved an objective response.
- 30% DCR; 3 patients achieved stable disease per RECIST criteria with tumor volume decrease of up to 17%

SLS009

Highly Innovative Next Generation CDK9 Inhibitor

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SLS009: Highly Specific and Selective Next Generation CDK9 Inhibitor

CDK9 (with Cyclin T1) forms Positive Transcription Elongation Factor b (P-TEFb) which enables transcription elongation of mRNA strands

P-TEFb is crucial for the survival of cancer cells, including the regulation of short-lived, anti-apoptotic survival proteins (MCL-1 and others) and oncogenes (c-MYC)
CDK9i can decrease MCL-1 and c-MYC levels, which could further induce apoptosis and cell cycle arrest

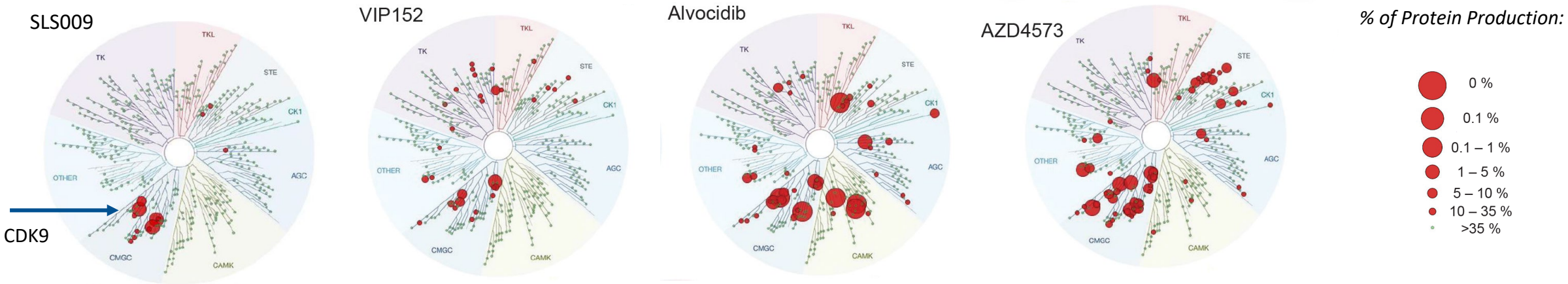
CDK9 activity is closely and negatively correlated with OS in a number of cancer types, including:

Hematologic cancers: AML and lymphomas
Solid cancers: breast cancer, osteosarcoma, pediatric soft tissue sarcomas, endometrial cancer, lung cancer, prostate cancer, melanoma and ovarian cancer

Next generation CDK9 inhibitors are more selective at lower doses

SLS009 has been designed in an effort to optimize the selectivity and potency: ~ 99.7% suppression of CDK9 and very limited observed off-target suppression

Kinome comparisons of select CDK9 inhibitors:



SLS009: Novel CDK9 Inhibitor for Transcriptional Driven Tumors

Broad applicability across cancer types with potential for both hematologic and solid cancers

Phase 2 Programs in Frontline and r/r AML

- 40 patients now enrolled in ongoing 80-patient Phase 2 trial in newly diagnosed AML patients, including those who become refractory early to AZA/VEN treatment identified through extensive transcriptomics, genomics, and proteomics models. Early topline data are expected in Q4 2026.
- Final data from completed Phase 2 study of SLS009 (tambiciclib) for the treatment of r/r AML met all primary endpoints and results from the trial to be presented at the 68th American Society of Hematology (ASH) Annual Meeting
  - Patients with relapsed/refractory monocytic AML achieved a 54% ORR with a median overall survival (mOS) of 5.8 months and patients with TP53-mutated AML achieved a 57% ORR and a mOS of 8.8 months, comparing favorably to historical outcomes in these adverse-risk settings of ~2.5 months

Phase 1 Clinical Trial in r/r Advanced Hematologic Malignancies

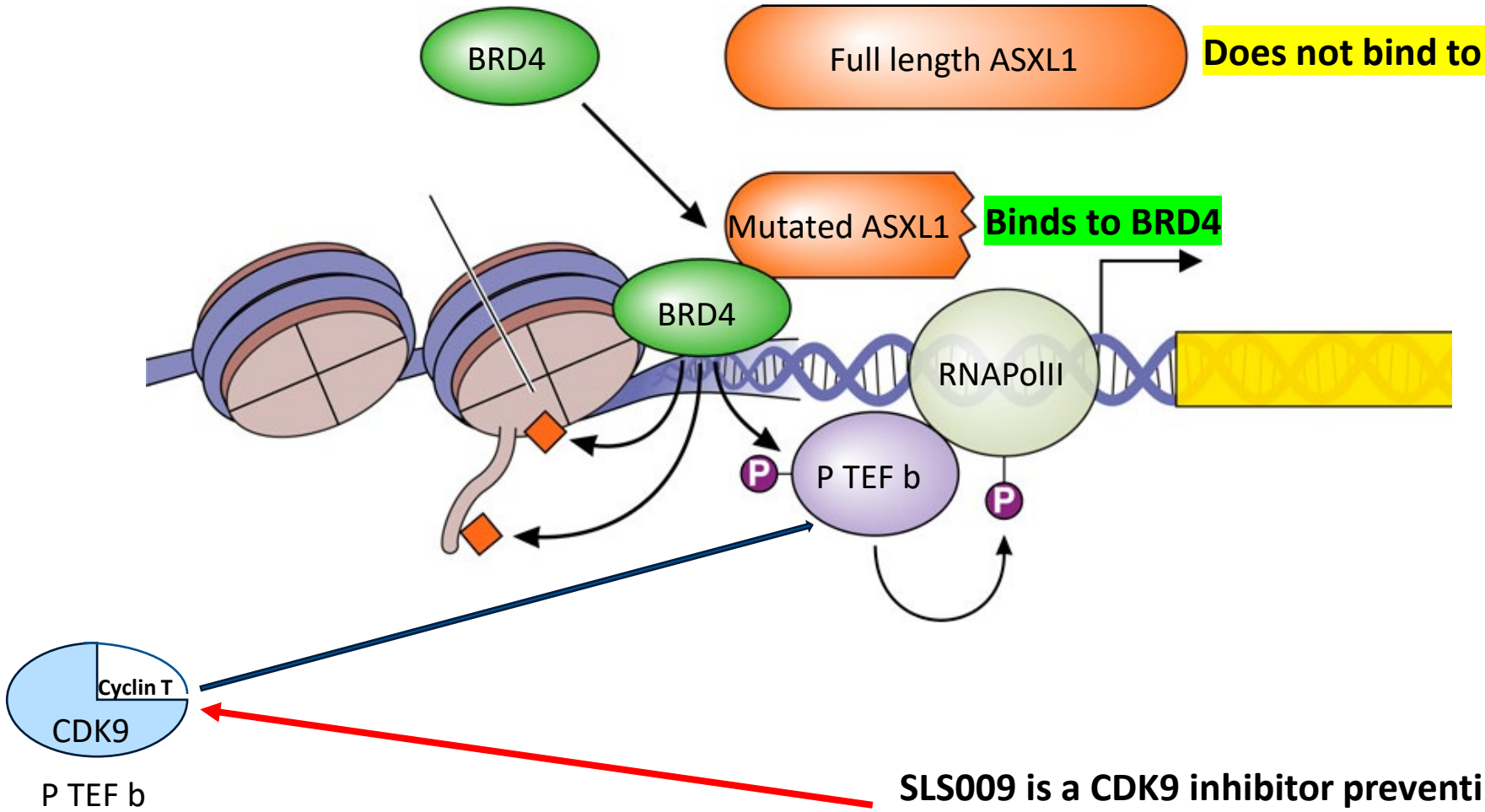
- Dose escalation completed in AML group; recommended phase 2 dose (RP2D): 60 mg
- Dose escalation completed in lymphoma group; RP2D: 100 mg

Preclinical Data; Preclinical NCI Sponsored PIVOT Program

- *In vitro* anti-proliferative activity and *in vivo* tumor reduction burden higher than other CDK9 inhibitors in development; potentially improved safety profile
- In September 2026, presented positive preclinical data presented at AACR 2026 Conference on Pancreatic Cancer: SLS009 demonstrated substantially greater single-agent activity than daraxonrasib in the daraxonrasib-resistant model, inducing 17.9% apoptosis versus 2.8% with daraxonrasib and 14.5% necrosis versus 2.7%
- PIVOT: highly selective program fully funded by NIH (no Company investment) using pediatric cancer cell lines and patient derived xenografts in vivo studies; output to include efficacy and safety data and genome/proteome/biomarker correlations
  - In May 2025, announced data for pediatric ALL* in 27 patient-derived ALL tumors. Median survival approximately tripled in the SLS009 arm compared to the vehicle control arm. SLS009 demonstrated delayed progression in 25/27 (93%) models and more than 2 times longer time to progression in 15/27 (56%) of ALL models.

*ALL - Acute lymphoblastic leukemia

SLS009 Mechanism of Action in ASXL1 Mutated AML



- Transcription speed is regulated by the BRD4 – PTEF b – RNAPolII axis
- ASXL1 frameshift mutations are a gain of function mutations
- C terminal truncated ASXL1 binds BRD4 and activates transcription of genes via pTEFb complex
- SLS009 inhibits CDK9 in the P TEF b thus disrupting increased transcription

SLS009 is a CDK9 inhibitor preventing assembly of CDK9 and Cyclin T into P TEF b

AML:
SLS009 and Aza/Ven Combination Preclinical Synergies

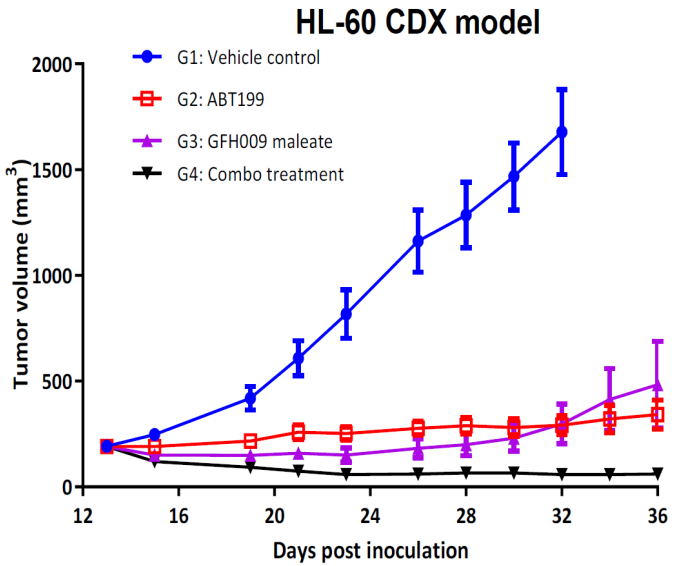
Main anti-apoptotic proteins in AML are BCL-2, MCL-1, and BCL-xL

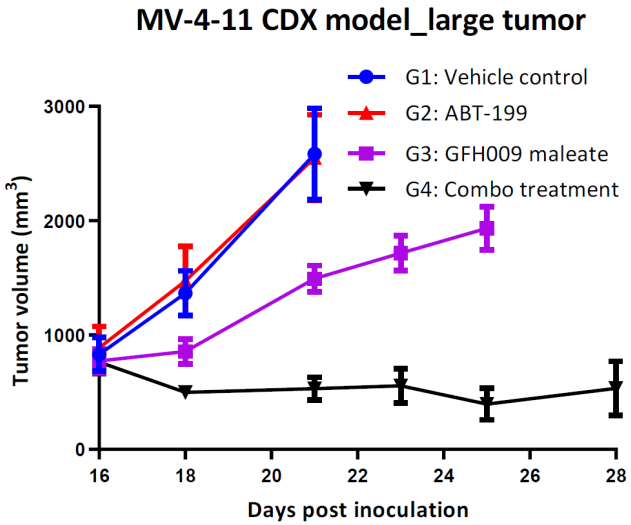
MCL-1 upregulation is considered to be the main mechanism of resistance to venetoclax (BCL-2 inhibitor)

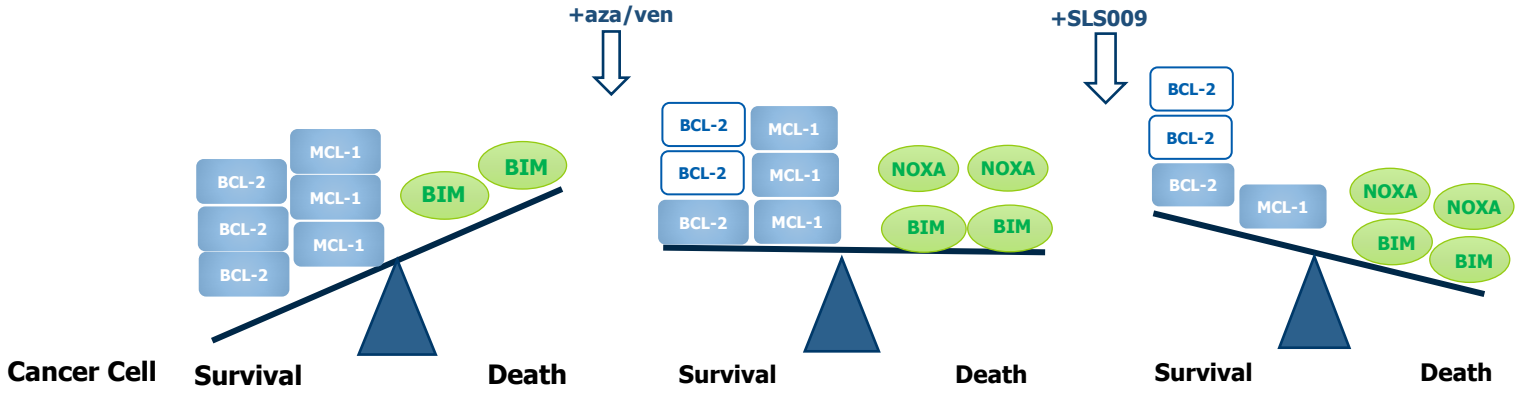
SLS009 suppresses production of MCL-1

Most leukemic cells are sensitive to combined suppression of BCL-2 and MCL-1

SLS009 synergy with aza/ven is not only due to BCL-2/MCL-1 combined inhibition but also due to NOXA generation caused by azacitidine







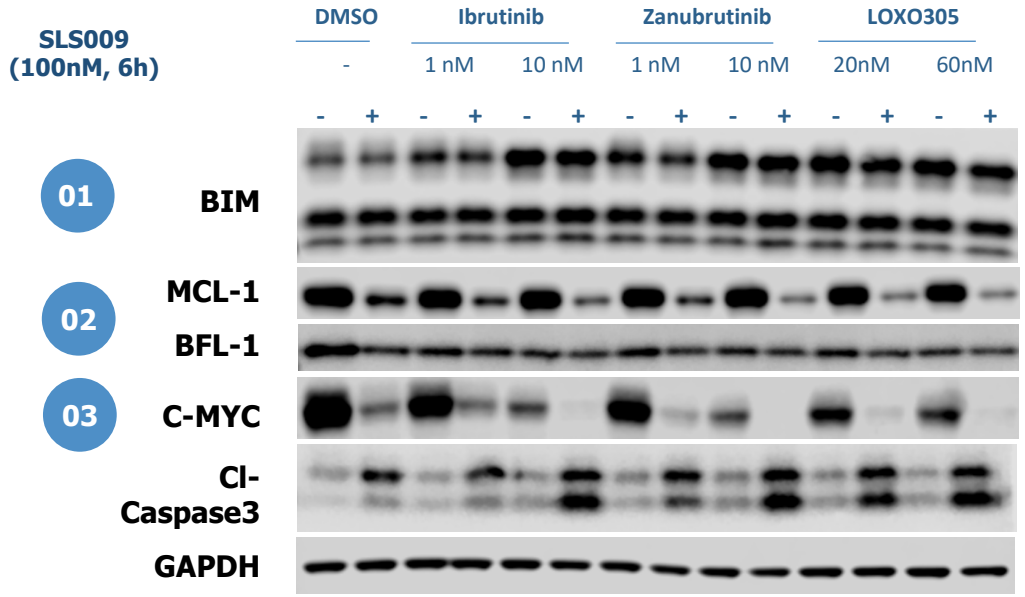
Lymphoma:

SLS009 and BTKi Combination Preclinical Synergies

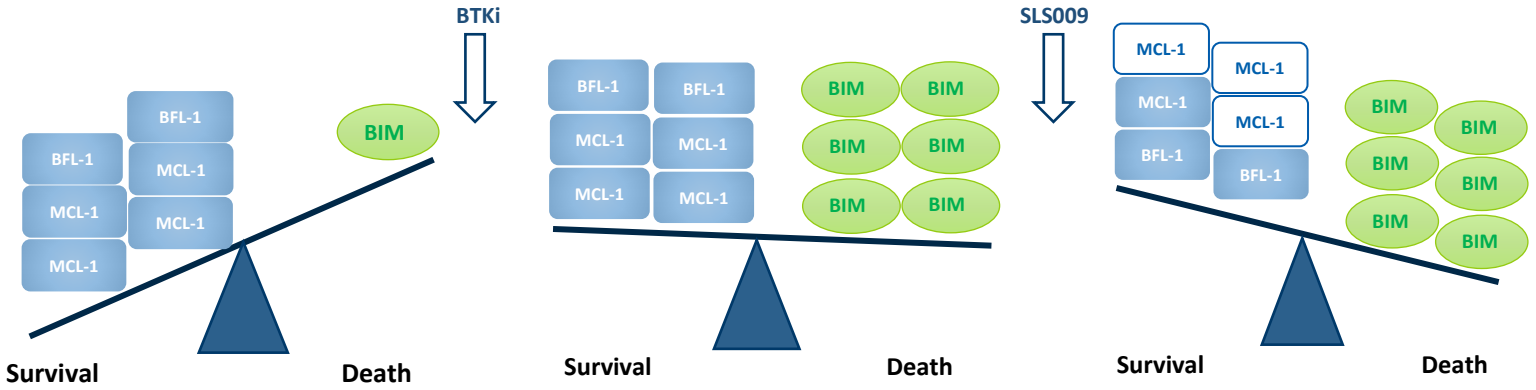
01 BTK inhibition primes diffuse large B cell lymphoma (DLBCL) cells for apoptosis by inducing BIM accumulation

02 SLS009 combination inhibits MCL-1 expression, triggering caspase-3 cleavage and cell death

03 The combination treatment also leads to enhanced suppression of C-MYC, a driver of DLBCL



- BTKi inhibitor alone (except DMSO as control), + SLS009 in combination with BTKi inhibitor, *OCI-LY10 cells were pre-treated for 24h with BTKis Ibrutinib, Zanubrutinib, or Loxo305, followed by co-treatment of SLS009 for 6h.



SLS009 + aza/ven combination P2a Trial in r/r AML – Trial Design

Target Population and Study Design

N = 62 patients

I/E criteria:

- Patients ≥ 18 yrs of age
- AML relapsed/refractory
- Refractory to or relapsed after a venetoclax containing regimen
- Peripheral WBC counts < 50K/µL
- ECOG PS 0-2

Study design:

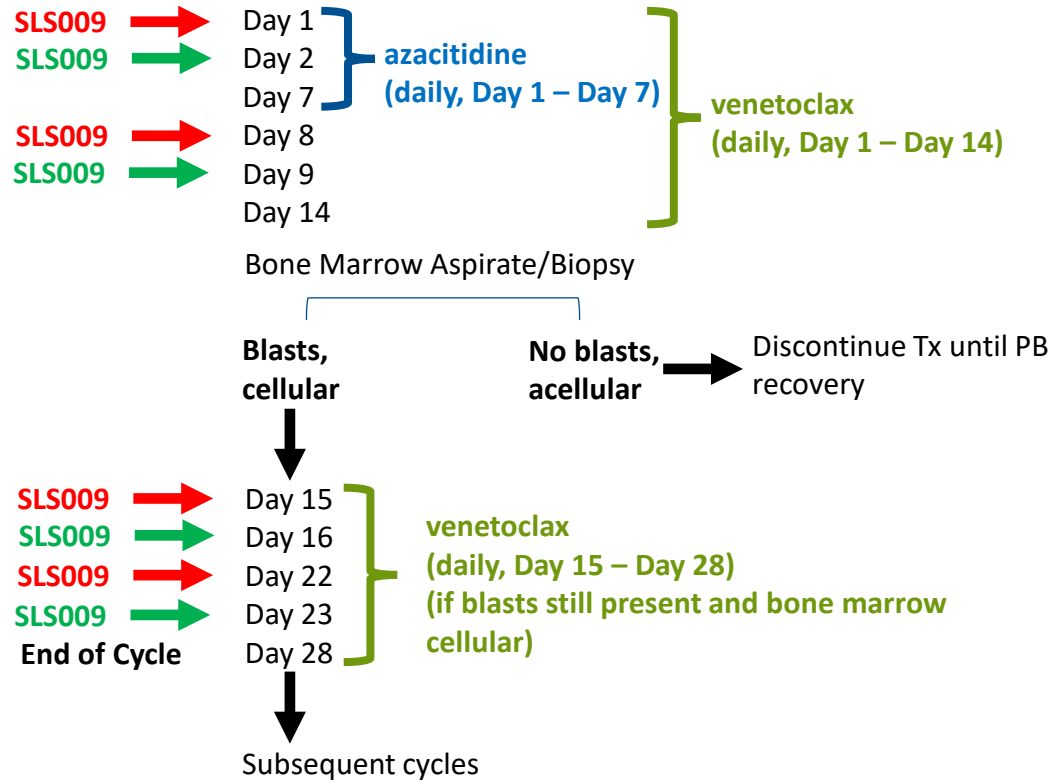
- Open Label
- Single Arm
- 60 mg DL randomization
- Multi-center P2a
- Combination therapy with:
- Azacitidine at 75 mg/m^2 and
- Venetoclax at 400 mg/day

Two SLS009 dose levels in two regimens:

- DL1 (safety): 45 mg
  - 45 mg QW (5-10 patients)
- DL2 (efficacy): 60 mg
  - 60 mg QW (5-10 patients)
  - 30 mg BIW (5-10 patients)
  - 30 mg BIW (10-15 patients with ASXL1 mutations)
  - 30 mg BIW (10-15 patients with other myelodysplasia-related mutations)

Administration and Treatment Algorithm

Initiated end of June 2023



Administration:
QW: 4-hour infusion
BIW: 30-minute infusion

Endpoints and Assessments

Primary Endpoints:

- Composite complete response rate (CRc)
- Duration of response (DOR)
- Safety and Tolerability of SLS009 in combination with aza/ven:
- Dose Limiting Toxicities (DLTs)
  Adverse events (AEs)
- The incidence and severity of all AEs

Secondary Endpoints:

- Event free survival (EFS)
- Overall Survival (OS)
- PK parameters for SLS009 and venetoclax in combination therapy: AUC0-t (Area under the plasma concentration-time curve (from zero to the time of the last measurable concentration))

Pharmacodynamic Assessments:

- MCL-1 levels
- C-MYC levels

SLS009 – All Primary Endpoints Met in Phase 2a Study

- Higher response rates and median overall survival observed in r/r AML patients receiving SLS009 + ven/aza therapy versus historical results in venetoclax-based regimens alone
- Safety profile: no dose-limiting toxicities (DLT) at any of the studied dose levels, and no treatment-related high-grade (≥G3) toxicities were observed
  - Hematologic toxicities profile was consistent with aza/ven standalone treatment
- Optimal dosing regimen for SLS009 determined at 30 mg BIW

Responses observed in AML MR, with superior ORR and survival compared to non-MR patients:

Dose	45 mg, QW	60 mg, QW	30 mg, BIW	All r/r AML cohorts	30 mg, BIW	30 mg, BIW
AML type	r/r AML	r/r AML	r/r AML		r/r AML w/ ASXL1 mutation	r/r AML MM
ORR	10%	33%	40%	34%	50%	50%

- Response rate of patients at the optimal 30 mg BIW dose (40%) far exceeds the targeted 20% benchmark
- Median Overall Survival (mOS) of 8.9 months in patients with r/r to venetoclax-based Tx AML MR and 8.8 mOS in all r/r to venetoclax-based Tx at a 30 mg BIW, with a median of 1 prior line of treatment, surpasses the historical benchmark of 2.4 months
- Additional data from the completed Phase 2 trial to be presented at ASH 2026, showing patients with r/r monocytic AML achieved a 54% ORR with a mOS of 5.8 months and patients with TP53-mutated AML achieved a 57% ORR and a mOS of 8.8 months, comparing favorably to historical outcomes in these adverse-risk settings of ~2.5 months

SLS009 – Front Line Trial Planned Following FDA Guidance

Following a productive end of Phase 2 meeting, the FDA recommended that SELLAS proceed into a trial to also include newly diagnosed, first-line AML patients eligible for venetoclax/ azacitidine (aza/ven) therapy, where the Agency believes clinical benefit might be greatest

- The randomized 80-patient trial has enrolled 40 patients to date and includes two groups of patients:
  - Predictive biomarker cohort: Newly diagnosed patients unlikely to benefit from standard aza/ven therapy based on molecular profiling
  - Early resistance cohort: Patients who initiate treatment with aza/ven but demonstrate a confirmed lack of response after the first two treatment cycles (standard aza/ven Tx is 4 cycles)
- This precision approach allows SELLAS to target subpopulations with high unmet need and greatest potential for benefit
- Study data may allow for a potential Accelerated Approval pathway and/or support New Drug Application
- Preliminary topline data (40 patients) expected in Q4 2026

About ASXL1 Mutations

ASXL1 mutations are associated with poor prognosis in myeloid diseases

- Lower response to the current treatment options
- Lower median survival
- Increased AML transformation for MDS

ASXL1 mutations in Hematologic Malignancies with ASXL1m Frequency ≥5%

Condition	ASXL1m Frequency	US Condition Incidence
AML (Acute Myeloid Leukemia)	20%	20,800
MDS (Myelodysplastic Syndrome)	20%	10,000
MPN (Myeloproliferative Neoplasms)	10%	20,000
CMML (Chronic Myelomonocytic Leukemia)	43%	1,100
	Total	**51,900**

ASXL1 in Solid Cancers with ASXL1m Frequency ≥5%

Condition	ASXL1m Frequency	US Condition Incidence
CRC MSI-high (Colorectal Cancer with High Microsatellite Instability)	55%	22,500
Cervical Ca. (invasive)	5%	13,800
Liver Ca.	10%	42,400
	Total	**78,700**

Two Clinical Stage Cancer Therapies

Compound	Indication	Combo agent	Preclinical	Phase 1	Phase 2	Phase 3	Anticipated Milestones
GPS	Maintenance in AML CR2/CRp2 (REGAL)						Positive topline results announced on November 17, 2026
	2nd/3rd Line Ovarian Cancer	pembrolizumab					Completed
	MPM	nivolumab					Completed
SLS009	r/r AML	venetoclax / azacitidine					ASH Presentation Q4 2026
	First-line AML	venetoclax / azacitidine					40 patients enrolled to date with preliminary topline data expected in Q4 2026
	Hematological Malignancies						Completed
	NIH PIVOT (Pediatric Tumors)						Completed Data announced 1H2025

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Thank you!

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