SELLAS Life Sciences Group

Corporate Presentation

February 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 20, 2025 and in its other filings with the Securities and Exchange Commission. 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.

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

- Phase 3 REGAL clinical trial for acute myeloid leukemia (AML) in patients achieving CR2: 126 patients
  - Successfully passed futility, safety, and efficacy review at interim analysis, demonstrating potential efficacy and merits continuation (January 2025)
  - Final analysis upon reaching 80 events; 72 events have occurred in the trial as of December 26, 2025
  - Phase 2 clinical trial for AML in patients achieving second complete remission (CR2): 21 vs 5.4 months in favor of GPS: p-value 0.02
- 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

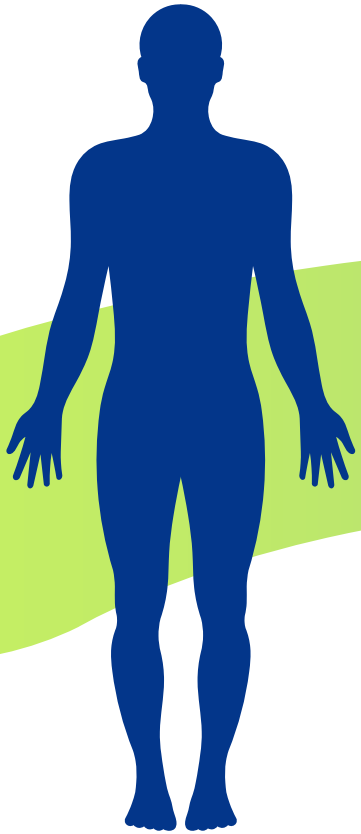
SLS009 (tambiciclib)

Novel Highly Selective CDK9 Inhibitor

- Phase 2 trial in combination with aza/ven in relapsed and/or refractory (r/r) AML: July 2025 announced all primary endpoints met:
  - Median Overall Survival (OS) 8.8 months at optimal dose level and 8.9 months in AML Myelodysplasia-Related Changes (AML MR) - expected mOS for r/r AML patients ~2.4 months
  - Overall Response Rate (ORR) of 41% at the 30mg BIW optimal dose, 44% in AML MR patients, and 50% in AML MR with Myelomonocytic/Myelomonoblastic (M4/M5) – all exceeding the prespecified target response rate of 20%
  - Identified biomarker, ASXL1 mutations, exhibited strong efficacy in r/r AML patients – predictive marker and showed strong efficacy in pre-clinical models (CRC, NSCLC, ALL)
- SLS009 in combination with aza/ven in newly diagnosed AML with high-risk features is planned for Q1 2026, with European enrollment anticipated in Q2 26
- ODD from FDA & EMA, Fast Track & RPDD from FDA
- More than ~150 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 to initiate 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

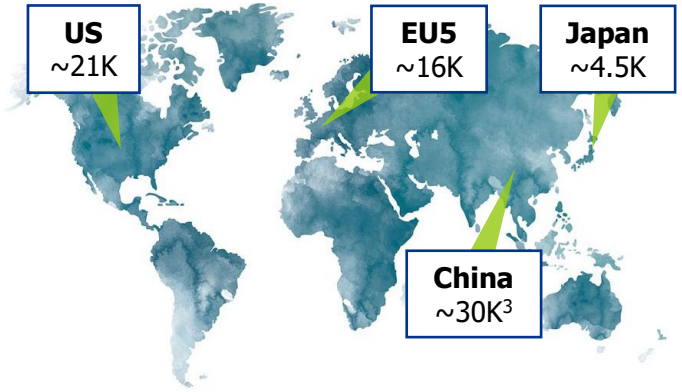
SLS009 in r/r AML
Ph2 initiated 2Q23

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

GPS Lead Indication
Ongoing Ph 3 in CR2 AML

CR2 population will 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

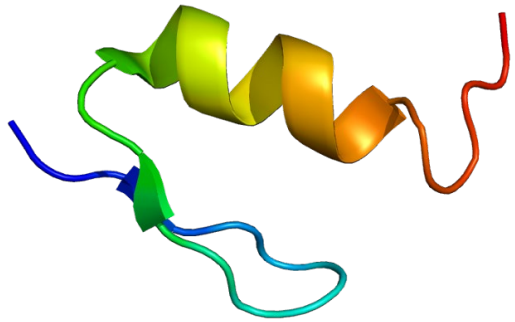
GPS Drug Candidate

- **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

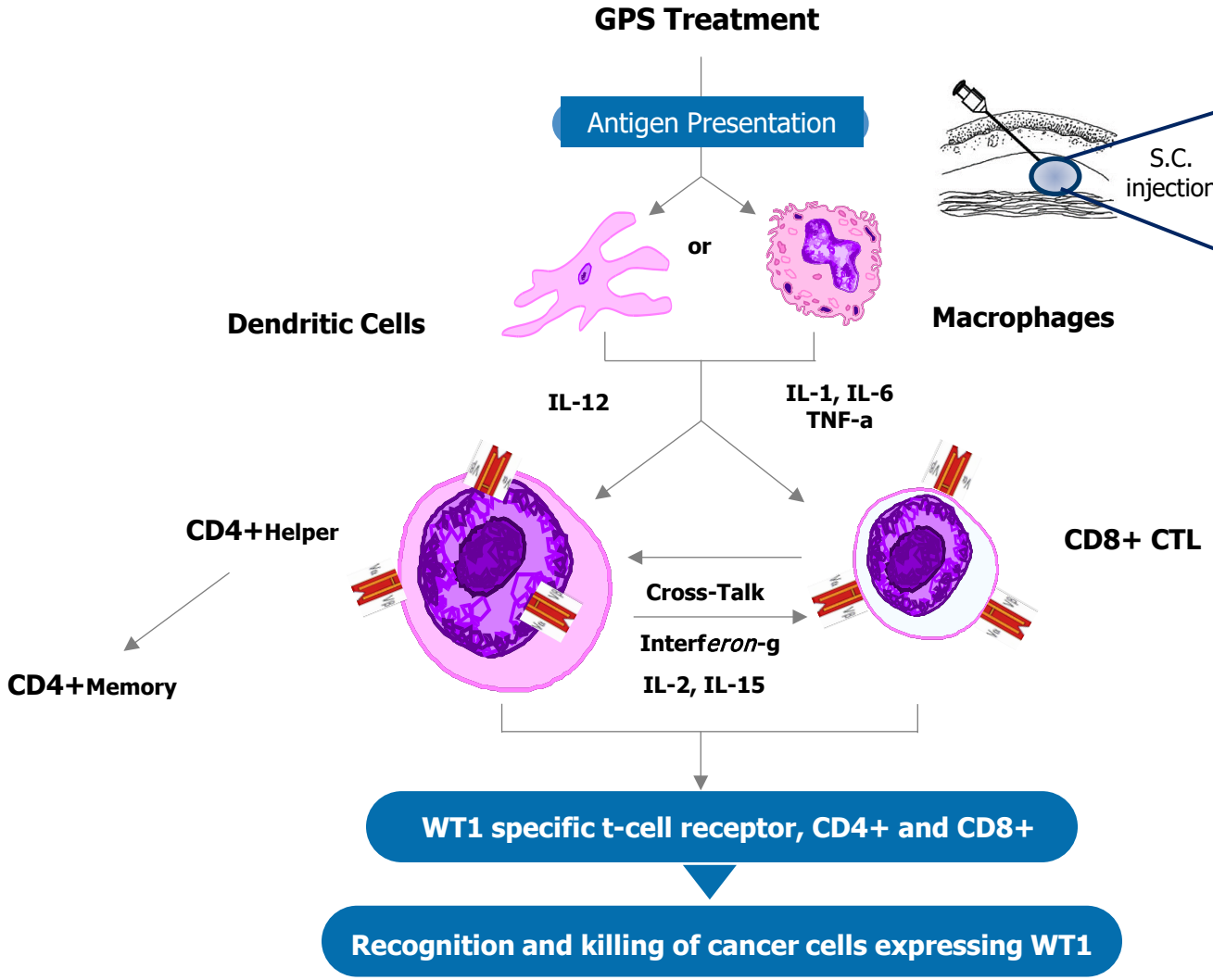
- WT1 ranked **#1 cancer antigen** by the NCI[1]
- Intracellular oncofetal antigen, WT1 not expressed in adult tissues, which **lowers potential off-target toxicity**
- Potential to treat **20+ hematological** and **solid tumor** cancers
- Densely and almost universally expressed in AML: **~97% express WT1**

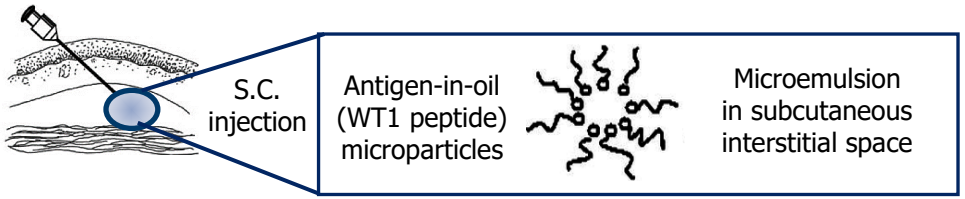
Wilms Tumor 1 Protein



[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%)

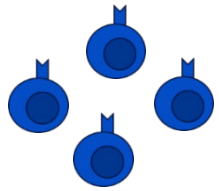
Sources: Gomez-Nunez M,et al. LeukRes.2006;30:1293-8; Pinilla-IbarzJ, et al. Leukemia. 2006;20:2025-33; SchijnsVE, et al. Curr. Protoc. Immunol. 2014;106:2.18.1-7.

GPS-Treated Lymphocytes Reactive to WT1 Protein

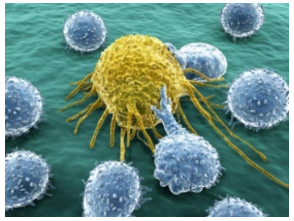
Epitope Spreading:

A Key Element for Cancer Immunotherapy

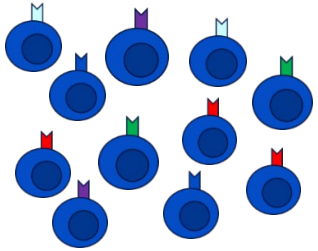
GPS-specific T-cells recognize and kill tumor cell



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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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

35.5 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%).

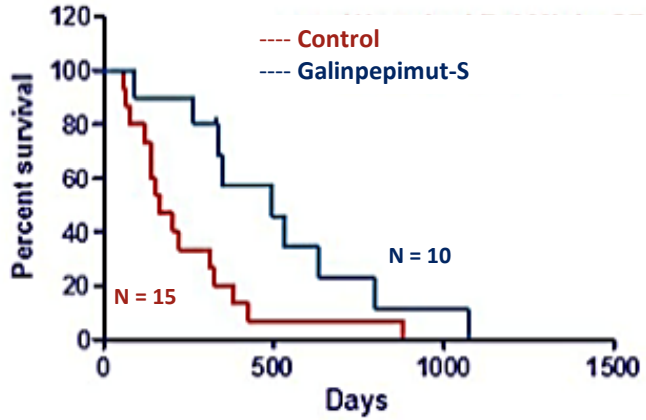
Survival Benefit Seen in AML Patients in CR2

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)

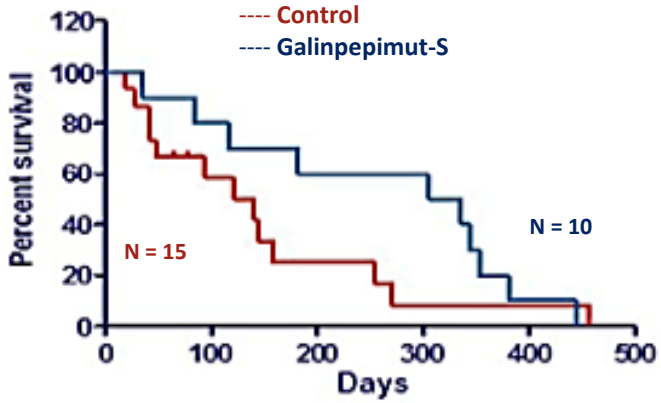
OVERALL SURVIVAL



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.

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

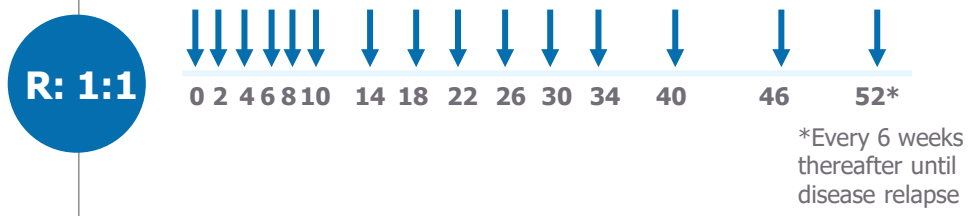
Treatment: Open Label, Randomized Multi-Center Phase 3 Trial

GPS

Montanide (500 µL/dose)

GM-CSF (70 µg/dose; d-2 & d0)

Schedule of Administration (weeks)



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)

Interim Analysis: January 2025

- The interim futility, efficacy, and safety analysis at 60 events demonstrates potential efficacy and merits continuation.
- GPS has shown preliminary signals of effectiveness, allowing the trial to advance toward completion.
- Median survival of over 13.5 months in the trial vs. historical median survival of 6 months for conventional therapy, as reported in a similar Phase 2 study. The next and final analysis will be conducted once 80 events (deaths) are reached.

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

GPS

Combination with Checkpoint Inhibitors

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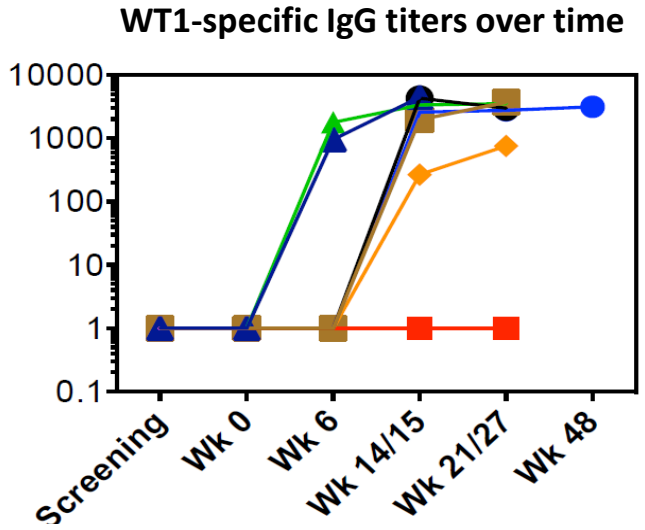
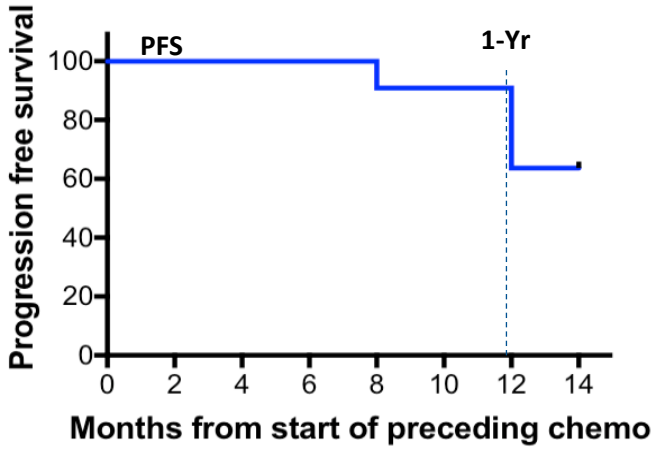
GPS Activity Demonstrated in Combo with Checkpoint Inhibitors in WT1 Expressing Solid Tumors

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 Topline data reported 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)

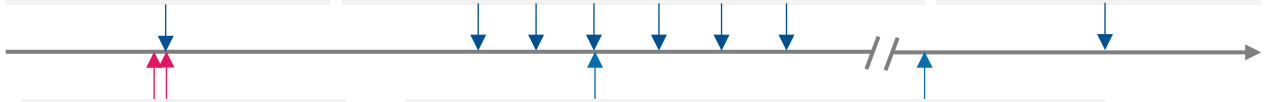
Metastatic or recurrent MPM

KPS ≥ 70%

WT1+ on immunohistochemistry (IHC)

1 prior course of pemetrexed-based chemo

Baseline biopsy ≤ 60 days before treatment starts



Weeks -2 and 0: GM-CSF (q2w)

Weeks 0 and 2: GPS (q2w)

Weeks 4,6,8,10,12,14: GPS + nivolumab (q2w)

Weeks 8 & 16: Imaging

Weeks 18: Repeat Biopsy

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%

3D Medicines License and REGAL Study Participation

Exclusive License Granted to 3D Medicines for Greater China in December 2020

Royalty-bearing license to develop, manufacture and commercialize GPS and heptavalent GPS (GPS-Plus)

All therapeutic and diagnostic uses

Greater China Territory: Mainland China, Hong Kong, Macau and Taiwan

Financial Terms

Upfront cash payment and milestones of $10.5 million received to date

Additional potential development, regulatory and commercial milestones totaling up to $191.5 million

Tiered royalties based upon percentage of annual net sales of GPS in Greater China (high single to low double digits)

Participation in REGAL: potential for accelerated entry into Greater China market

Accelerates timing of regulatory and commercialization milestones

Phase 3 REGAL study participation: $13 million milestone payments

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

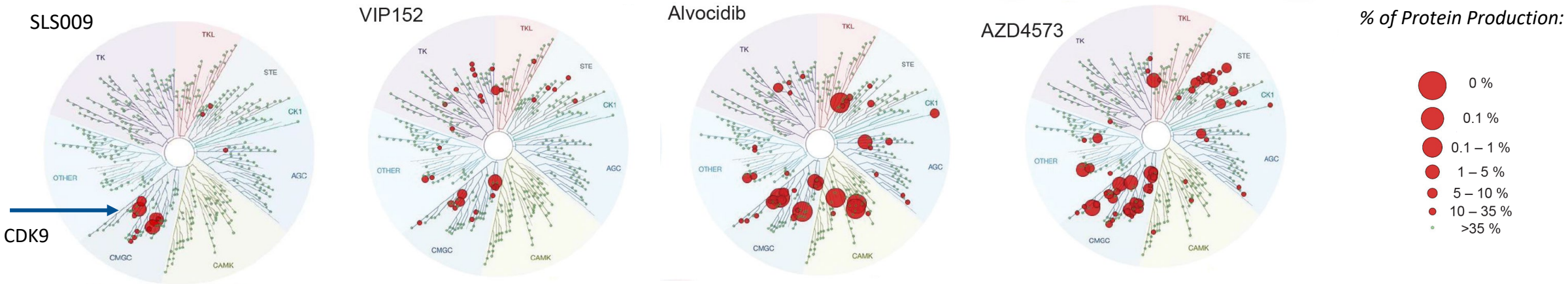
CDK9 activity is closely and negatively correlated with OS in a number of cancer types, including:

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 the optimal combination of selectivity and potency: 95% suppression of CDK9 and very limited 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

- Phase 2 open-label, single-arm, multi-center study in AML to evaluate safety, tolerability, and efficacy at two dose levels, 45 mg and 60 mg (60 mg QW or 30 mg BIW) with additional two cohorts evaluating the optimal dose of 30 mg BIW in r/rAML patients with ASXL1 mutations and r/r AML patients with other AML myelodysplasia-related changes in combination with aza/ven; completed in Q2 2024. The trial met all primary endpoints.
- Phase 2 study expansion showed 44% ORR across all r/r AML-MR cohorts and 58% ORR in pts with one prior line of therapy (ASH 2025)
- Identified biomarker, ASXL1 mutations, demonstrated strong efficacy in r/r AML patients
- FDA recommended advancement into first-line AML patient cohorts – enrollment expected to begin in Q1 26 in US and Q2 26 in Europe

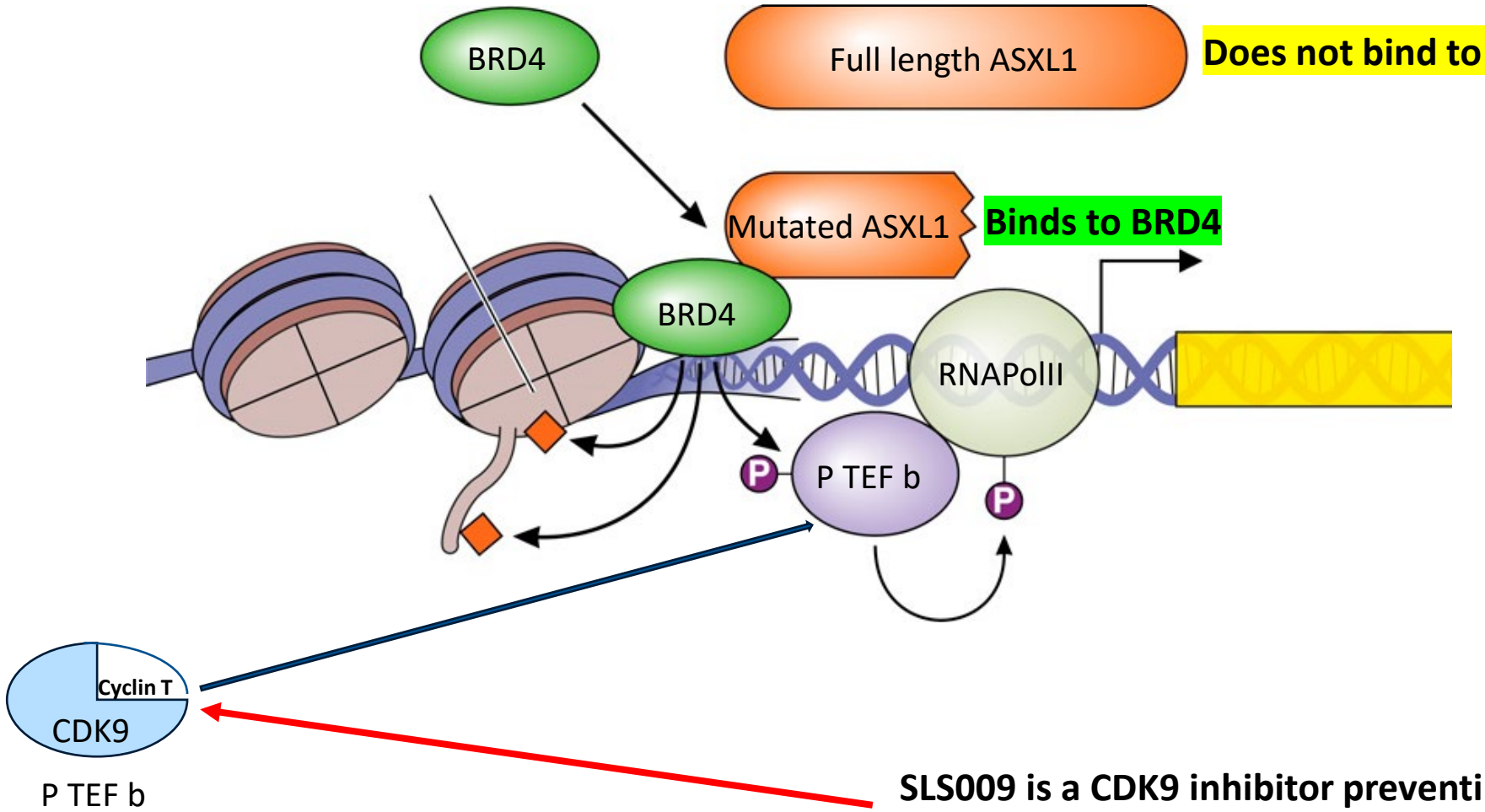
Phase 1 Clinical Trial in r/r Advanced Hematologic Malignancies

- 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
- 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 acute lymphoblastic leukemia (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.

SLS009 Mechanism of Action in ASXL1 Mutated AML



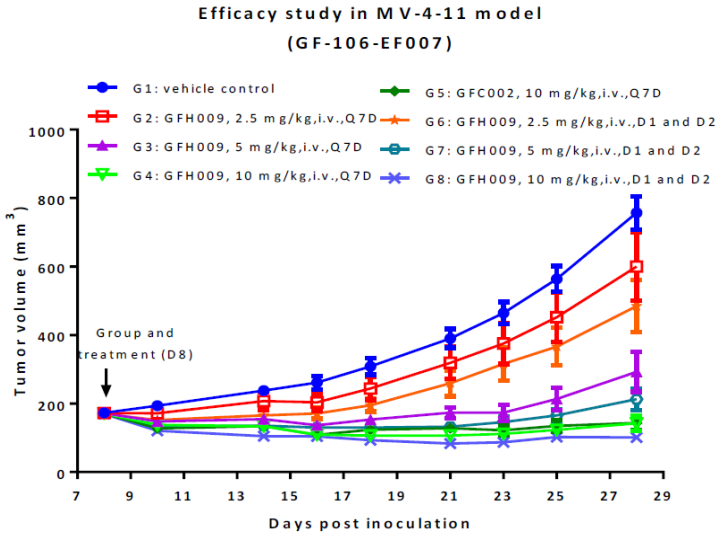
- Transcription speed is regulated by the BRD4 – PTEF b – RNAPolII axis
- ASXL1 frameshift mutations are a gain of function mutations
- C terminal truncated ASXL1 binds BRD4 and activates transcription of genes via pTEFb complex
- SLS009 inhibits CDK9 in the P TEF b thus disrupting increased transcription

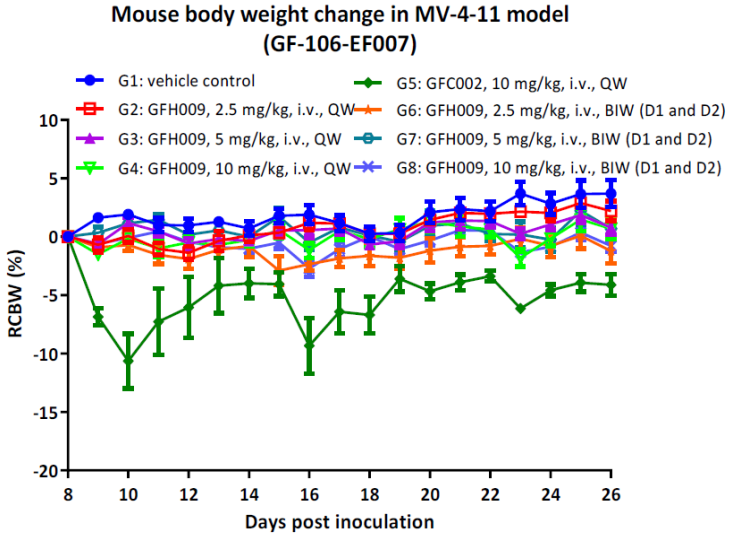
SLS009 is a CDK9 inhibitor preventing assembly of CDK9 and Cyclin T into P TEF b

Clinical Stage Selective CDK9 inhibitors: Preclinical Comparison

Preclinical data demonstrate potential for superior safety and efficacy compared to a key IV CDK9 inhibitor in development

Cell lines	SLS009 IC50 (72h)	VIP152 (formerly BAY-125112) IC50 (72h)
AML	4.8 ~33 nM	15.9 ~136 nM
Lymphoma	10.6~77.9 nM	16.6 ~138 nM
MM	33.6 ~151 nM	51.4 ~397 nM
ALL	13.4~35.7 nM	42.3 ~68.6 nM
CLL	25 nM	40.7 nM





Significantly lower doses of SLS009 achieve tumor inhibition compared to a key competing CDK9 inhibitor in vitro

In vivo, SLS009 had more anti-tumor effect than a key competing CDK9 inhibitor

Safety profile for SLS009 at a highly efficacious dose level was indistinguishable from the control, while administration of a competing CDK9i resulted in severe toxicity with >10% mouse body weight loss

GFC002 is a code for VIP152 (formerly BAY-1251152)

AML:
SLS009 and Aza/Ven Combination Preclinical Synergies

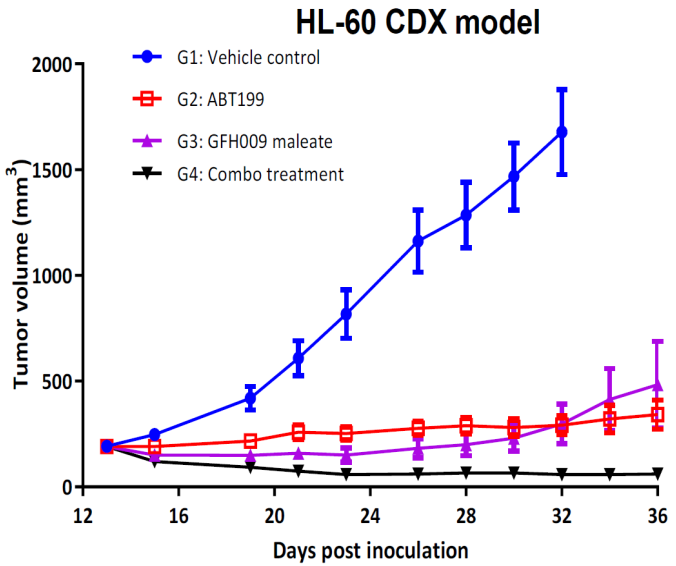
Main anti-apoptotic proteins in AML are BCL-2, MCL-1, and BCL-xL

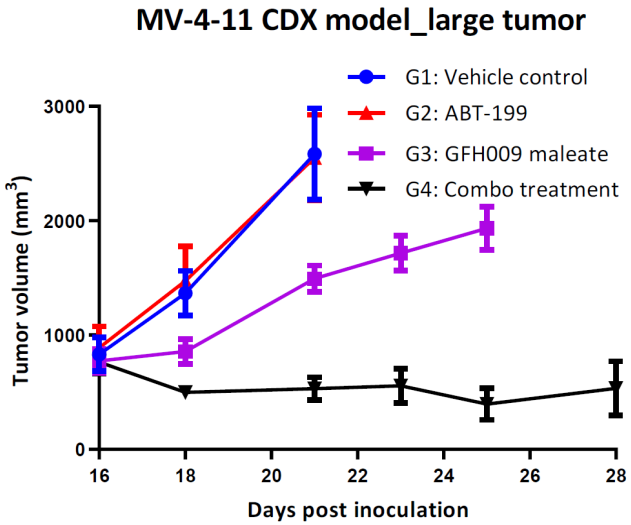
MCL-1 upregulation is considered to be the main mechanism of resistance to venetoclax (BCL-2 inhibitor)

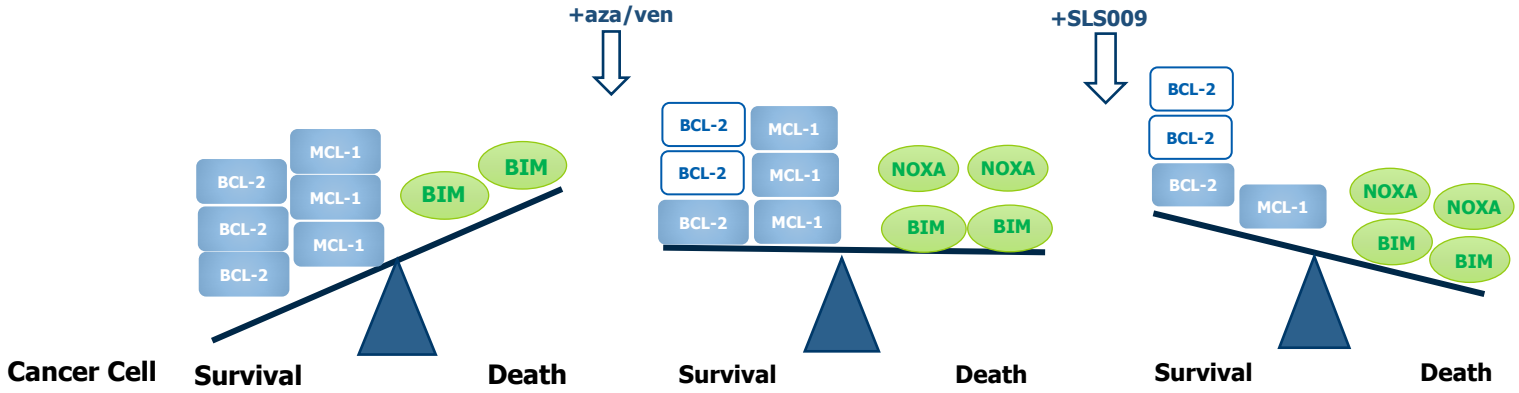
SLS009 suppresses production of MCL-1

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







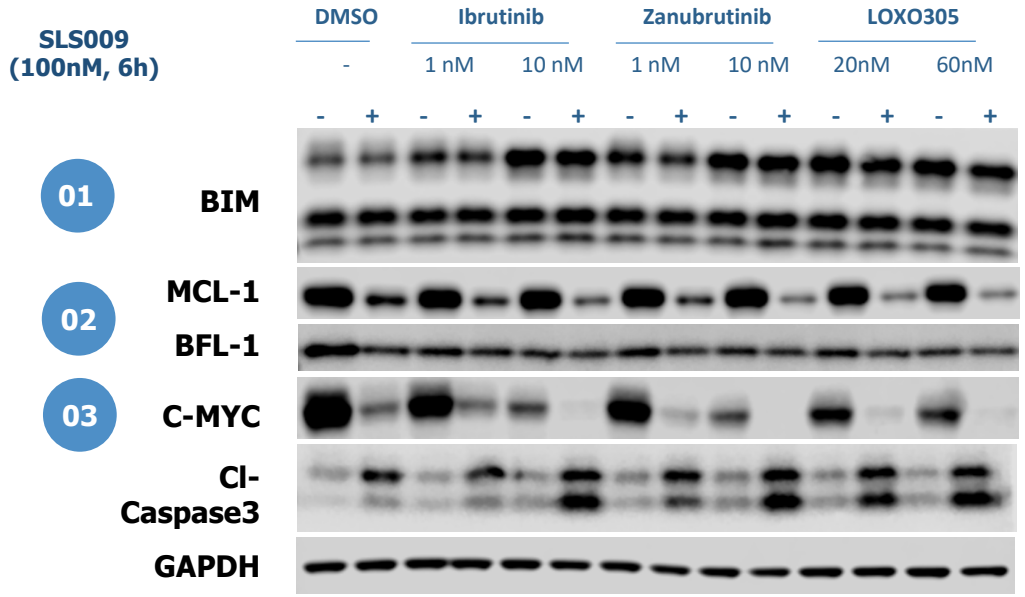
Lymphoma:

SLS009 and BTKi Combination Preclinical Synergies

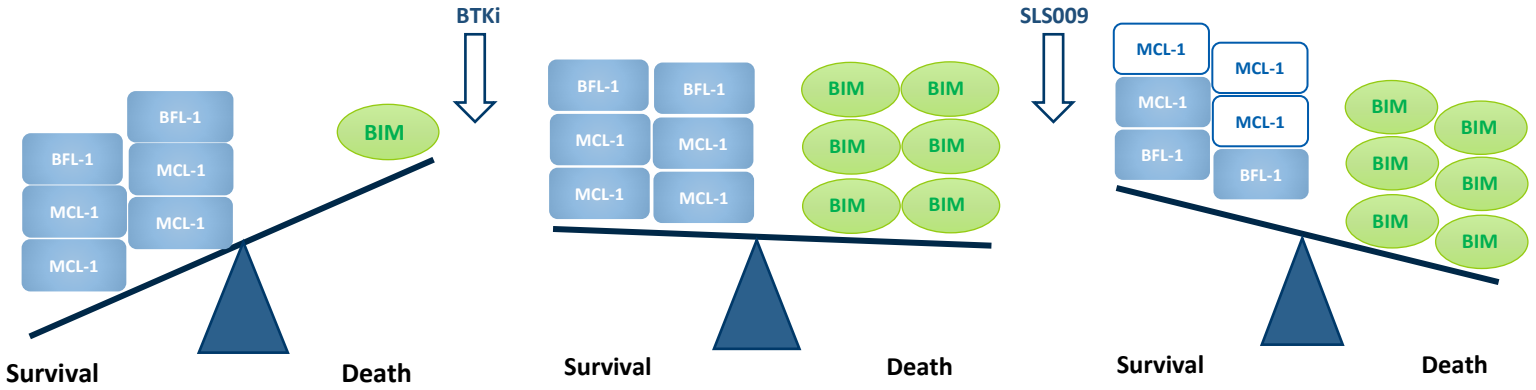
01 BTK inhibition primes diffuse large B cell lymphoma (DLBCL) cells for apoptosis by inducing BIM accumulation

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 Clinical Development

Phase 1 Trial r/r hematologic cancers

Target Population and Study Design

N = Up to 80 patients

Patients ≥ 18 yrs of age
Relapsed/refractory hematologic cancers:
CLL/SLL, lymphoma or AML
≥ 2 prior therapies
ECOG PS 0-2

Open Label
Single Arm
Monotherapy
Multi-center

Administration and Dose Escalation Algorithm

Administration Schedule: BIW or QW 21 days cycles, continued until intolerable toxicities or progression of disease

Additional dose level added for lymphoma (completed):

- SLS009 100 mg QW
- SLS009 75 mg QW

Completed enrollment

- SLS009 60 mg QW
- SLS009 45 mg QW
- SLS009 30 mg BIW
- SLS009 40 mg BIW
- SLS009 22.5mg BIW
- SLS009 30 mg QW
- SLS009 15 mg BIW
- SLS009 9 mg BIW
- SLS009 4.5 mg BIW
- SLS009 2.5 mg BIW

Endpoints and Assessments

Primary Endpoints:
Safety and Tolerability of SLS009:
Dose Limiting Toxicities (DLTs) Time Frame: 21 days (The incidence of DLTs)
Safety and Tolerability of SLS009: adverse events (AEs) Time Frame: approximately 2 years
The incidence and severity of all AEs

Secondary Endpoints:

PK parameter AUC0-t (Area under the plasma concentration-time curve (from zero to the time of the last measurable concentration))
Time Frame: approximately 3 months

PK parameter AUC0-∞ (Area under the plasma concentration-time curve (from zero to infinity)
Time Frame: approximately 3 months

Pharmacodynamic Assessments:

MCL-1 levels
C-MYC levels
(% change during treatment)

SLS009 Biological and Clinical Activity in Phase 1

AML Patients

- 9 mg BIW: -50.0% BMB reduction at week 4, pretreatment blast burden 40.0%
- 15 mg BIW: -53.8% BMB reduction at week 4, pretreatment blast burden 45.5%
- 30 mg QW: -57.1% BMB reduction, pretreatment blast burden 7.0%
  - CR achieved at week 12, durable response for 8 months, and patient alive for 16 months per last follow-up*
- 45 mg QW: - 61.3% BMB reduction, pretreatment blast burden 15.5%
- 60 mg QW: - 77.3% BMB reduction at week 4, pretreatment blast burden 66.0%
- RP2D: 60 mg QW

Lymphoma Patients

- 52 patients enrolled, among evaluable patients:
- Responses Observed Across Dose Levels
  - 14.7% Clinical Response Rate Overall
  - 35.3% Overall Disease Control Rate
  - 36.4% Clinical Response Rate in PTCL Patients
- Decrease in MCL1 and/or MYC Biomarkers Observed in 100% of Patients in a Dose-Dependent Manner in Once Per Week Administration Regimen
- RP2D: 100 mg QW

Safety Summary*

No DLTs in AML, including neutropenia related

In lymphoma, 1 DLT at 100 mg, G3 neutropenia

I No drug-related deaths I No significant off-target toxicities

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

Target Population and Study Design

N = Up to 60 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

SLS009 → Day 1
SLS009 → Day 2
Day 7
azacitidine (daily, Day 1 – Day 7)
SLS009 → Day 8
SLS009 → Day 9
Day 14
venetoclax (daily, Day 1 – Day 14)

Bone Marrow Aspirate/Biopsy

Blasts, cellular → SLS009 → Day 15, SLS009 → Day 16, SLS009 → Day 22, SLS009 → Day 23, End of Cycle Day 28

venetoclax (daily, Day 15 – Day 28) (if blasts still present and bone marrow cellular)

No blasts, acellular → Discontinue Tx until PB recovery

Subsequent cycles

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 2 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
- Excellent 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%	41%	34%	50%	50%

- Response rate of patients at the optimal 30 mg BIW dose 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 presented at ASH 2025 showed a 44% ORR across all AML-MR cohorts treated at 30 mg BIW, and a 58% ORR in patients with one prior line of therapy

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 unlikely to benefit from venetoclax/ azacitidine (aza/ven) therapy, where the Agency believes clinical benefit might be greatest

- The randomized 80-patient trial is currently in preparation and is expected to begin enrollment in Q1 2026. The trial will include two groups:
  - 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
- U.S. enrollment anticipated in Q1 2026 and European enrollment anticipated in Q2 2026 in collaboration with IMPACT-AML

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 with Minimal Competition

Compound	Indication	Combo agent	Preclinical	Phase 1	Phase 2	Phase 3	Anticipated Milestones
GPS	Maintenance in AML CR2/CRp2 (REGAL)						Interim Analysis (60 events): 1Q'25 As of 12/26/25: 72 events Final Analysis following 80th event
	2nd/3rd Line Ovarian Cancer	pembrolizumab					Completed
	MPM	nivolumab					Completed
SLS009	r/r AML	venetoclax / azacitidine					Final Data Announced on July 15, 2025
	First-line AML	venetoclax / azacitidine	Planned				Enrollment to begin by Q1 2026 in US and Q2 2026 in Europe
	Hematological Malignancies						Completed
	NIH PIVOT (Pediatric Tumors)						Completed Data announced 1H2025

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Thank you!

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