SELLAS LIFE SCIENCES GROUP

Company Overview

NASDAQ: SLS

November 2022

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, but are not limited to, statements related to the potential of our clinical candidates as therapeutic options for various cancers, the general development of the Company's product candidate pipeline and anticipated milestone dates, and the effects of the Company's approach to cancer treatment. 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 the COVID-19 pandemic and its impact on the Company's clinical plans and business strategy, immune-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 31, 2022 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.

Overview

SELLAS is a late-stage clinical biopharmaceutical company focused on the development of **novel cancer therapies** for a broad range of indications

- **Galinpepimut-S (GPS):** highly novel and engineered immunotherapy targeting the **Wilms Tumor 1 (WT1) antigen**
  - Discovered and licensed from **Memorial Sloan Kettering Cancer Center**
  - **Ongoing Phase 3 pivotal trial (REGAL)** for acute myeloid leukemia (AML) in patients achieving second complete remission (CR2)
  - **Phase 1/2 combination trials with anti-PD1 drugs** - collaboration agreement with Merck (KEYTRUDA®) in advanced ovarian cancer and an IST study with Memorial Sloan Kettering Cancer Center and Bristol Myers Squibb (OPDIVO®) in metastatic pleural mesothelioma (MPM)

- **GFH009**: in-license of U.S. and ROW (other than Greater China) rights to highly selective CDK9 inhibitor from GenFleet Therapeutics (Shanghai) Inc.
  - Potentially first and best in class differentiated small molecule
  - Phase 1 clinical trial ongoing in China and United States for advanced hematological malignancies
  - Phase 2 studies in relapsed and/or refractory (R/R) AML and certain solid tumors, respectively, expected to commence in United States by the end of Q1 2023
  - Potential treatment for pediatric cancers

Experienced Management Team and Board of Directors

Management Team	Position	Prior Experience/Affiliations
 **Angelos Stergiou, M.D., ScD h.c.**	President, CEO & Board Director	PAION, Biovest International, ACCENTIA BIOPHARMACEUTICALS, Analytica INTERNATIONAL, anavex LIFE SCIENCES Corp.
 **Dragan Cicic, M.D.**	SVP, Clinical Development	KLUS PHARMA, Actinium Pharmaceuticals, Inc.
 **Barbara A. Wood**	EVP, General Counsel & Corporate Secretary	OPHTHOTECH, (osi) pharmaceuticals
 **Robert Francomano**	SVP, Chief Commercial Officer	Stemline, Baxalta, Pfizer, gsk, AstraZeneca
 **John T. Burns, CPA**	SVP, Finance & Chief Accounting Officer	GALENA BIOPHARMA, MOSSADAMS

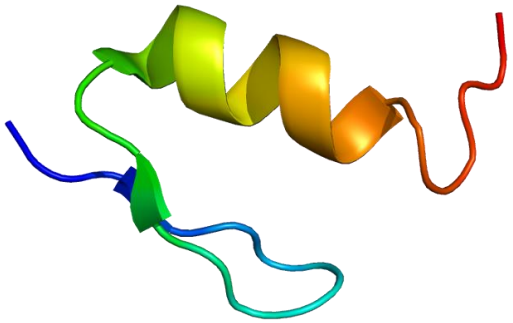
Board of Directors [1]	Position	Prior Experience/Affiliations
 **Jane Wasman**	Board Chair, Nom & Corp Gov Committee Chair	ACORDA THERAPEUTICS, Schering-Plough
 **John Varian**	Audit Committee Chair	XOMA, ARYx THERAPEUTICS, VERSARTIS
 **Robert Van Nostrand**	Compensation Committee Chair	(osi) pharmaceuticals, ACHILLION, Intra-Cellular Therapies, Yield10 BIOSCIENCE
 **David Scheinberg, M.D., Ph.D.**	Science Committee Chair	Memorial Sloan Kettering Cancer Center, Progenics Pharmaceuticals
 **Katherine Bach Kalin**	Board Member	GENFIT TOWARDS BETTER MEDICINE, Brown ADVISORY Thoughtful Investing., FemHealth Ventures, Celgene

1. Dr. Stergiou is also a member of The Board of Directors.

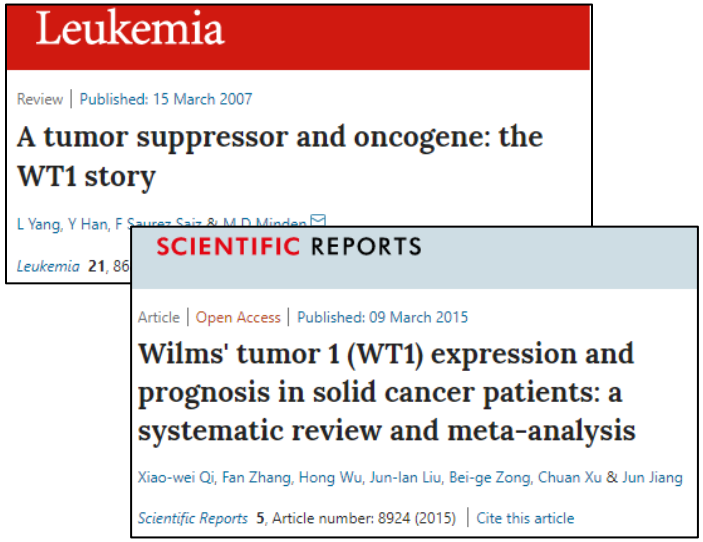
Scientific Advisory Board – World Renowned Experts in Immunotherapy And Oncology

Name	Position
 **Jeffrey Weber, M.D., Ph.D. - Chair**	Deputy Director of the Perlmutter Cancer Center, Co-director of the Melanoma Research Program at the New York University (NYU)-Langone Cancer Center
 **Jedd D. Wolchok, M.D., Ph.D.**	Chief, Melanoma & Immunotherapeutics Service, Memorial Sloan Kettering Cancer Center (MSKCC)
 **Alexander M.M. Eggermont, M.D.**	Chief Scientific Officer, Princess Maxima Center for Pediatric Oncology, Utrecht, NL Former Director General, Institut Gustave-Roussy Cancer Campus - Grand Paris, Villejuif, FR
 **Larry W. Kwak, M.D., Ph.D.**	Vice President and Deputy Director of the City of Hope Comprehensive Cancer Center; Associate Director Cancer Center Translational Research & Developmental Therapeutics for the City of Hope National Medical Center
 **Javier Pinilla-Ibarz, M.D.**	Director of Immunotherapy for Malignant Hematology at the H. Lee Moffitt Cancer Center
 **Sattva Neelapu, M.D., Ph.D.**	Professor and Deputy Department Chair at the Department of Lymphoma and Myeloma, Division of Cancer Medicine, The University of Texas MD Anderson Cancer Center
 **Guenther Koehne, M.D., Ph.D.**	Chief, Bone Marrow Transplantation and Hematologic Oncology, Miami Cancer Institute

WT1: Nuclear Transcription Factor with Oncogenic Properties



Wilms Tumor 1 Protein



Top ranked cancer antigen by the **National Cancer Institute** (NCI)[1]

Potential to treat **20 or more** cancer types creates a **large potential market** for WT1 targeting therapies

Broadly detectable in AML, where it is densely and **almost universally expressed (97%)**. It is also **expressed in multiple solid tumors** and **cancer stem cells**

Highly expressed and presented in cancer cells, enabling recognition and killing by specifically immunized T-cells

Intracellular oncofetal antigen, WT1 **not found appreciably in adult tissues**, which **lowers potential off-target toxicity**

Does **not** down-regulate or become mutated frequently

Sources: Gaiger, Leukemia. 1998; 2. Dao & Scheinberg, Best Pract Res Clin Haematol. 2008; 3. Nishida & Sugiyama, Methods Mol Biol. 2016; NED: no evidence of disease
Notes: 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.

Galinpepimut-S (GPS): Working to Change the Face of Immunotherapy

✓ Pivotal Phase 3 Trial Underway in CR2 AML Patients (REGAL)

- Monotherapy **AML maintenance** after second complete remission (CR2)
- Interim read-out currently expected in **late 2023 or early 2024**
- Final read-out currently expected **by end of 2024**
- **Orphan Drug Designation** (US and EU) and **Fast Track status**
- Phase 2 results:
  - GPS reaching **21.0** months median overall survival vs. **5.4** months standard of care ($p < 0.02$).

✓ Validated Wilms Tumor 1 (WT1) Target

- #1 ranked cancer antigen by the NCI
- GPS targets tumors that overexpress the **WT1 protein**

✓ Innovative Technology Exclusively Licensed from Memorial Sloan Kettering

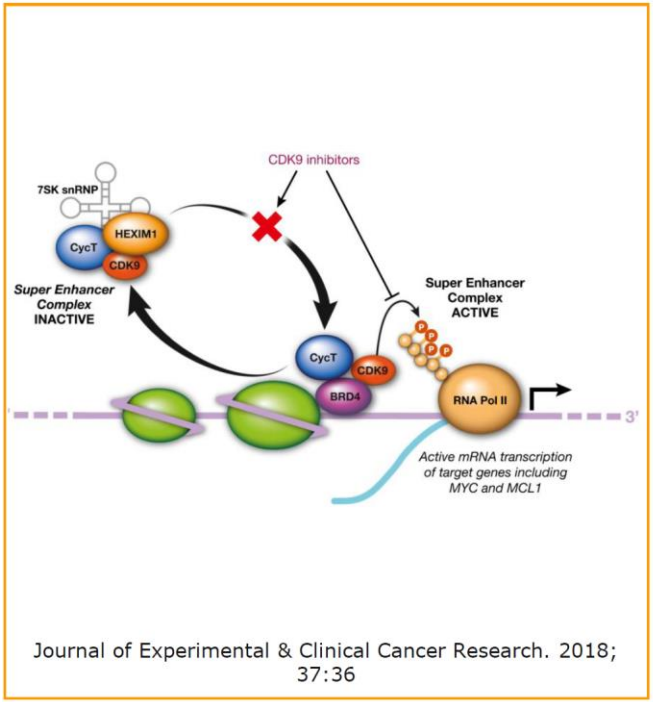
- Highly engineered immunotherapy designed to boost immunogenicity and break tolerance
- Applicable to the majority (**~90%**) of human **HLA types**
- Activation of **both CD4+** (memory) and **CD8+** (tumor killing) cells
- Sustained remission

✓ Broad Applicability in I/O Treatment Setting

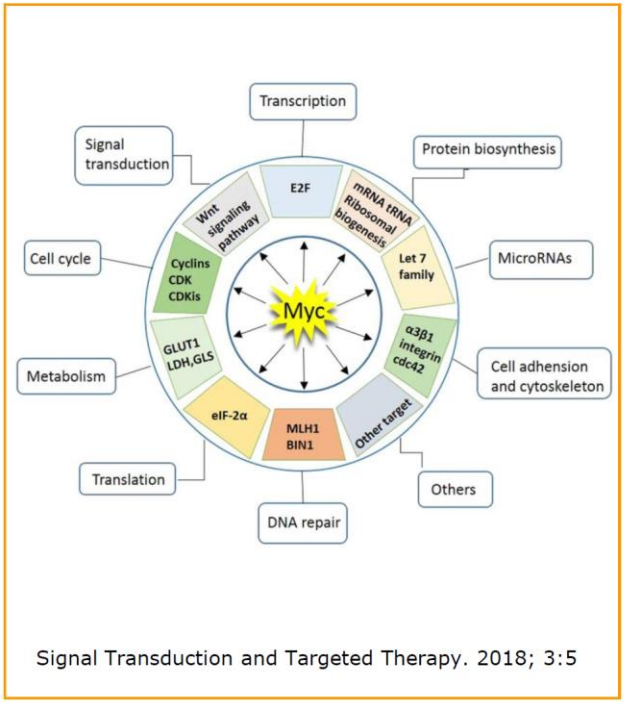
- Potential to be used as **monotherapy or in combination** with other I/O agents in multiple **solid and hematological** tumor types; two studies of GPS in combination with PD1 inhibitors ongoing/recently completed
- Composition of matter patent to at least **2033**

CDK9: Major Driver Across Variety of Cancers

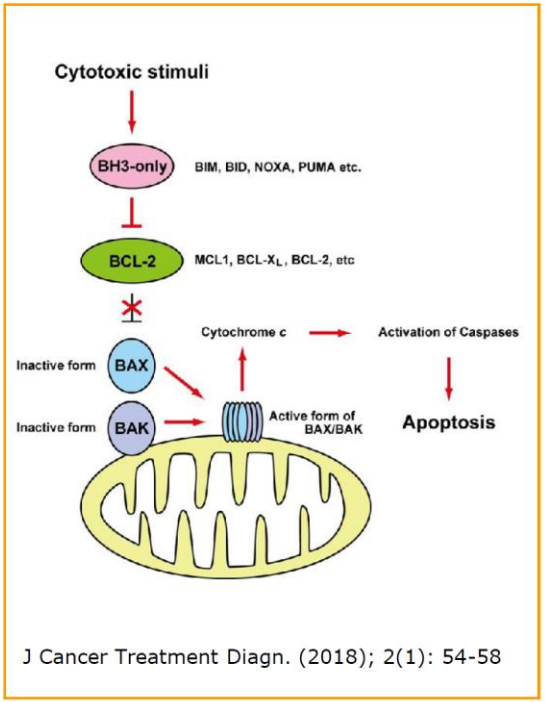
CDK9 promotes multiple cell cycle and survival mechanisms, disruption of which can drive tumor cell killing



Journal of Experimental & Clinical Cancer Research. 2018; 37:36



Signal Transduction and Targeted Therapy. 2018; 3:5



J Cancer Treatment Diagn. (2018); 2(1): 54-58

- CDK9/Cyclin T1 phosphorylates RNA pol II (Ser2 of the CTD), promoting transcription elongation and mRNA maturation
- Regulates short-lived, anti-apoptotic survival proteins (MCL-1 and others) and oncogenes (c-MYC)
- CDK9i can decrease MCL-1 and c-MYC level, which could further induce apoptosis and cell cycle arrest

GFH009: Next Generation Novel CDK9 Inhibitor

ΣELLAS LIFE SCIENCES GROUP



✓ **Phase 1 Trial underway in United States and China in R/R advanced hematologic malignancies**

- Monotherapy dose escalating trial
- Phase 1 trial **completion expected by the end of 2022**
- Two Phase 2 trials **expected to commence by end of Q1 2023**



✓ **CDK9: target known for crucial role in transcriptional elongation driven tumors**

- Target well understood but, until recently, compounds lacked specificity
- GFH009 has been observed in preclinical studies to have high specificity for CDK9 and low affinity for other CDK family targets
- Potential for biomarker driven development and basket trials



✓ **Cutting edge compound with limited competition and high combination potential**

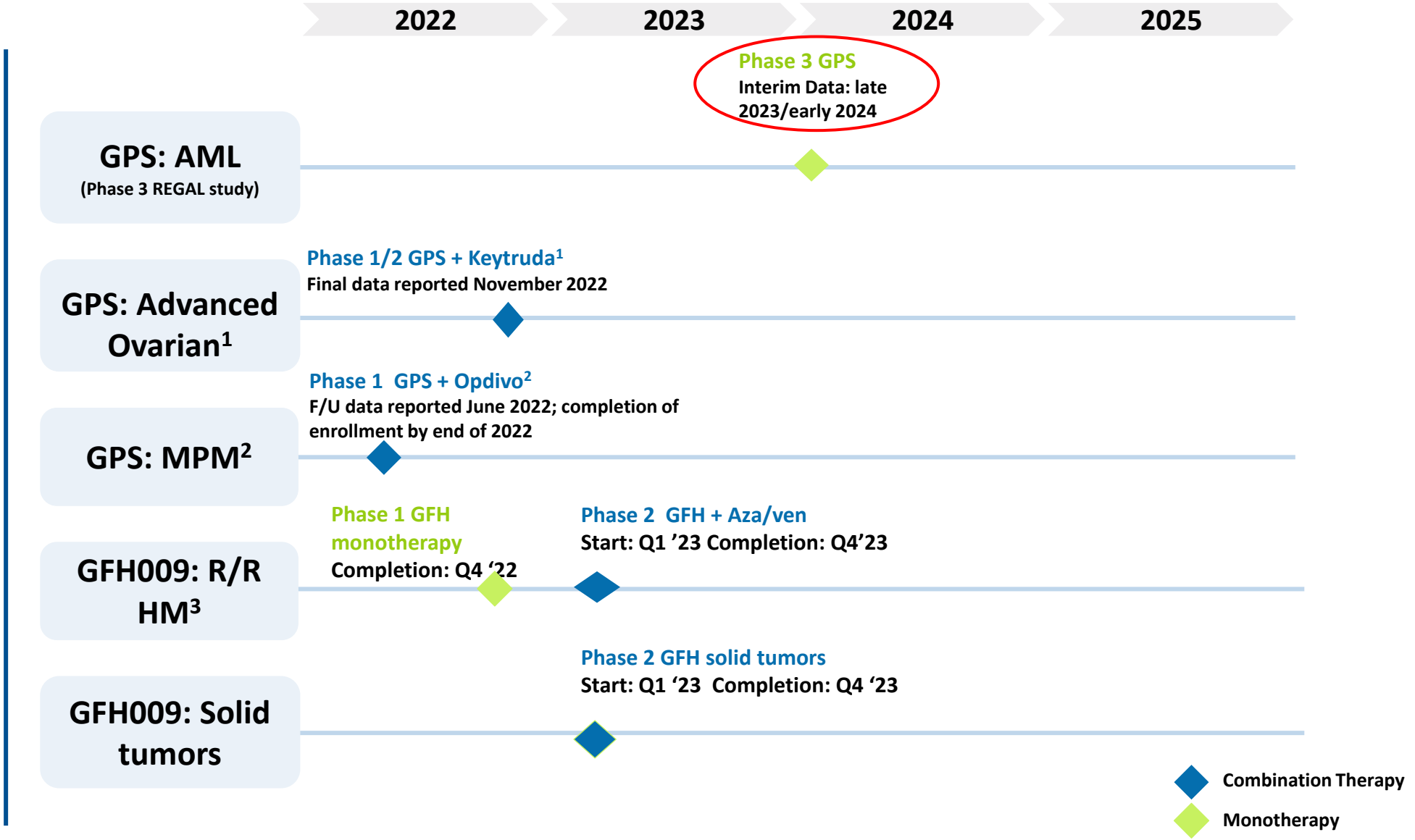
- Only small number of compounds in the same class in clinical development
- High combination potential with both new and established cancer drugs



✓ **Broad Applicability across cancer types**

- Potential for both hematologic and solid cancers
- Potential for pediatric cancers
  - **Potential for rare pediatric disease priority review voucher**

Upcoming Milestones



Notes: 1. Merck and Bristol-Myers Squibb supply study drugs but do not have any rights to GPS. 2. Malignant Pleural Mesothelioma (MPM).
3. R/R HM: relapsed and/or refractory hematologic malignancies



GPS – Mechanism of Action

ΣELLAS
LIFE SCIENCES GROUP

Differentiated Immunotherapy Using Heteroclitic Technology

'Heteroclitic' peptides are engineered to have an artificially introduced single point amino acid (AA) sequence mutation: (i) peptide binds with a higher affinity, (ii) breaks tolerance to the native sequence and (iii) stimulates stronger T cell response

GPS

"Heteroclitic" multivalent peptide chains addressing 25 WT1 epitopes

- 4 peptides of variable lengths (9-22 amino acids)
- 2 native sequences
- 2 synthetic sequences with single AA mutations

Peptide sequences (position)	
WT1-A1: *YMFPNAPYL (126–134)	9-mer
427 long: RSDELVRHHNMHQRNMTKL (427–445)	19-mer
331 long: PGCNKRYFKLSHLQMHSRKHTG (331–352)	22-mer
122A1 long: SGQA*YMFPNAPYLPSCLES (122–140)	19-mer

We believe heteroclitic peptides increase immune response. Clinical data has shown:

- Synthetic sequence binds with higher strength (affinity) to the HLA molecules, generating stronger immune response
- Increases chance of **generating clonal T-cell population with recognition of at least one peptide**, and a clinical response against the tumor
- **Breaks tolerance to the native tumor sequence**
- Generates a response against the native sequence found in the tumor cells
- Spurs **multi-epitope, broad cross-reactivity** along the full length of the WT1 protein

Production of both CD4 and CD8 WT1-specific activated cells

- Native sequences designed to activate $CD4^+$
- Synthetic sequences designed to active $CD4^+$ and $CD8^+$

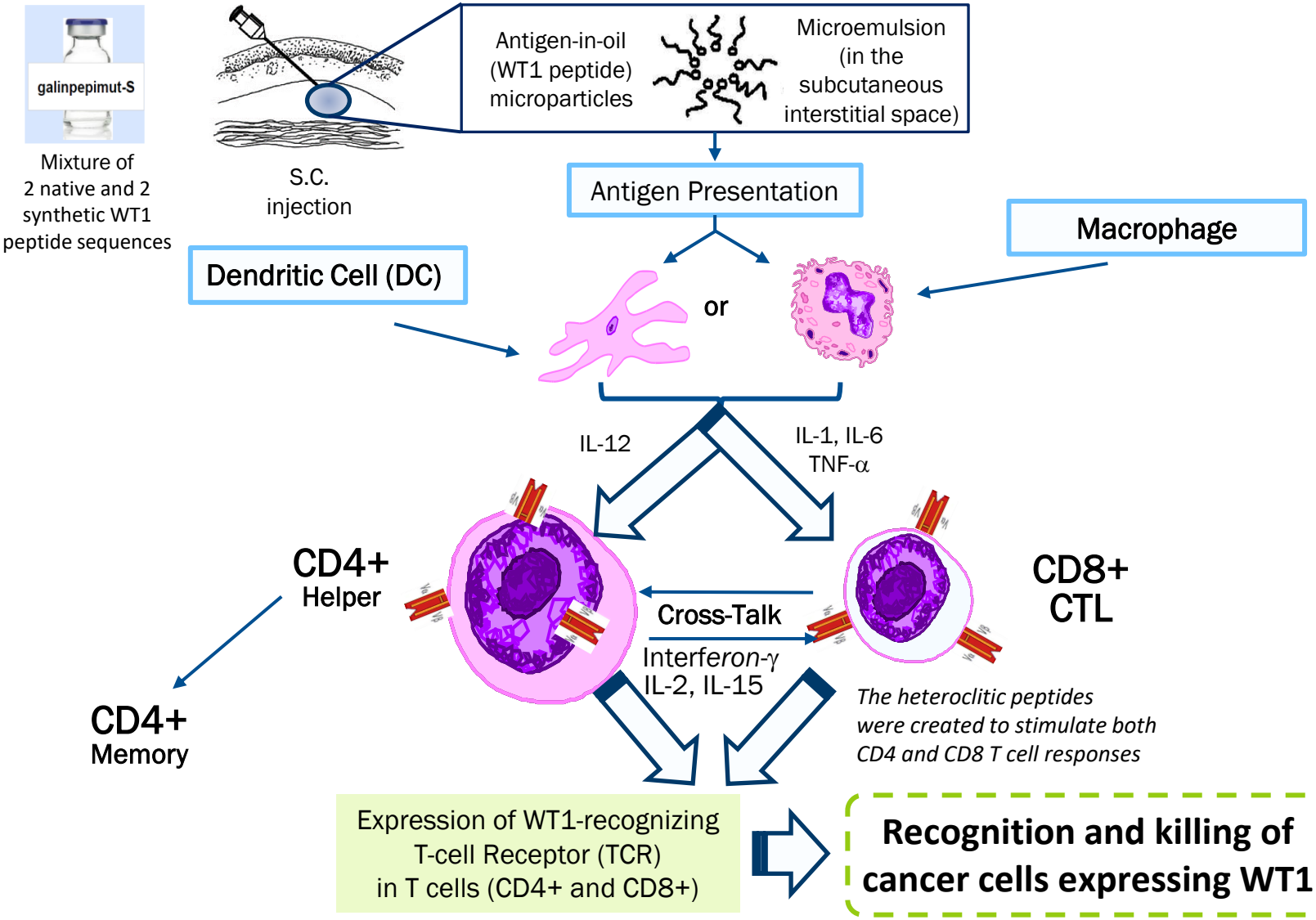
Specificity across multiple HLA types[1]

- Broad coverage of human HLA allelic variation
- Allows for 'off the shelf' subcutaneous injection use and lyophilized formulation

Sources: Pinilla-Ibarz, Leukemia 2006; Gomez-Nunez, Leuk Res, 2006; May, Clin Cancer Res 2007; Maslak, Blood, 2010; Krug, 2010 I-O: immuno-oncology.
Notes: 1. Predicted to be reactive with 10 MHC-type I [HLA-A & -B] and 15 MHC-Type II [HLA-DRB1-XX] molecules.

GPS Mechanism of Action

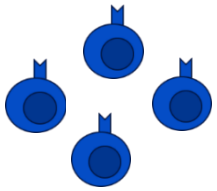
GPS's unique mechanism of action promotes a WT1-specific CD4 and CD8 T cell response



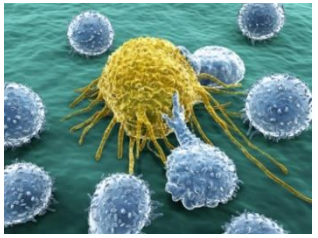
Sources: Gomez-Nunez M,et al. LeukRes.2006;30:1293-8; Pinilla-IbarzJ, et al. Leukemia. 2006;20:2025-33; SchijnsVE, et al. Curr. Protoc. Immunol. 2014;106:2.18.1-7.

Epitope Spreading:
A Key Element for Cancer Immunotherapy

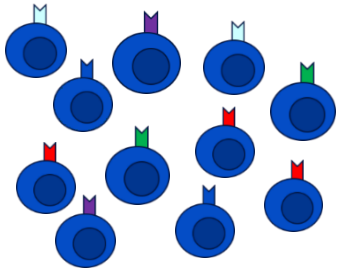
GPS-specific T-cells recognize and kill the tumor cell



Tumor cell death releases new antigens into the tumor microenvironment



New antigens cause creation of T-cells specific for a broader set of antigens vs which the host was not initially immunized against

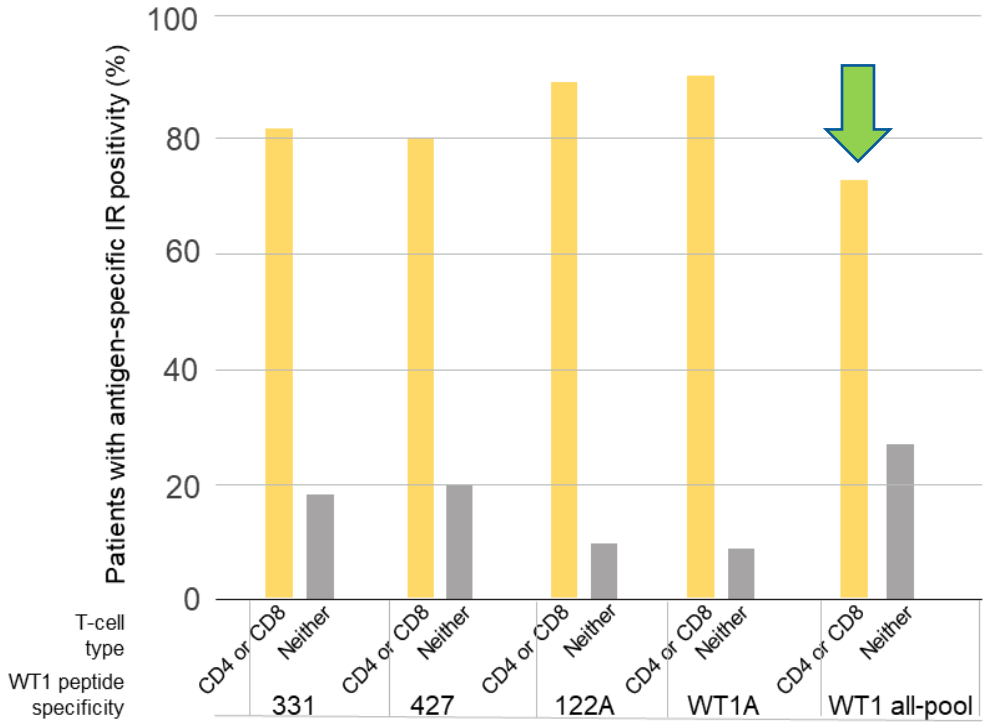


- Reactivity of lymphocytes from multiple myeloma (MM) patients treated with galinpepimut-S (GPS) tested against a collection of overlapping peptide epitopes of the entire WT1 protein (~113 pentadecapeptide fragments; ~230 epitopes; "WT1 – All pool")
- For CD8+, exposure of patient cells to these peptides at baseline observed to not trigger interferon responses – therefore, no WT1-specific CD8 cells exist in the host prior to GPS administration
- After GPS treatment, observed not only GPS peptide-specific CD8+ T cells, but also CD8+ (and CD4+) **T-cells reactive to WT1 epitopes for which the host was not specifically immunized**
- For this to happen, we believe there would have had to be **active cancer cell killing, release and presentation of WT1 peptide fragments, processing of additional WT1 epitopes by the immune synapse, and production of a broader, *de novo* expanded, repertoire of CD8+ clones specific to epitopes not included in GPS**

High Frequency of WT1-Specific Immune Responses - CD8 or CD4 - and Epitope Spreading in MM Patients in the GPS Phase 2 Trial

- Up to 91% of MM patients post-GPS therapy show antigen-specific T-cell (CD8 or CD4) immune response against at least one of the constituent WT1 peptides
- Evidence of epitope spreading (green arrow on graph on the right)
  - High frequency (83.3%) of T-cell responses to an 'all-pool' mixture of WT1-derived antigens (~240 epitopes) – against which patients were not specifically inoculated
- A distinctive link was observed between achievement of CR/VGPR clinical effect and frequency & potency of WT1-specific immune responses at the time point of completion of GPS therapy

Rates of antigen-specific IR positivity (CD8/CD4) at the time of completion of GPS therapy



CR: complete response; VGPR: very good partial response (degree of MM clinical responses, assessed based on criteria published by Kumar S, et al. Lancet Oncol. 2016)

GPS – Targeting AML

CR1 Phase 2 Trial Results

Phase 2 Open-label Clinical Trial in AML CR1 Patients

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

67.6 months vs 17.5 - 25 months

Overall Survival (>60 Years Patients):**GPS** vs **SOC**

35.5 months vs 9.5 - 16.8 months

Key Points:

- Pre-specified primary endpoint of 3 year OS > 34% was met in 47% of patients. AML historical OS 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 (statistically significant), 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+ or CD8+; 64% had a persistent, mainly CD4+ response at both early and late time points
- CD4+ responses seen across all HLA-Class II subtypes tested

Source: CR1 Phase 2 study - Maslak et al., Blood Adv. 2018. Notes: 1. In cohorts of pts 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

Completed Phase 2 Open-label Clinical Trial in AML CR2 Patients at Moffitt Cancer Center

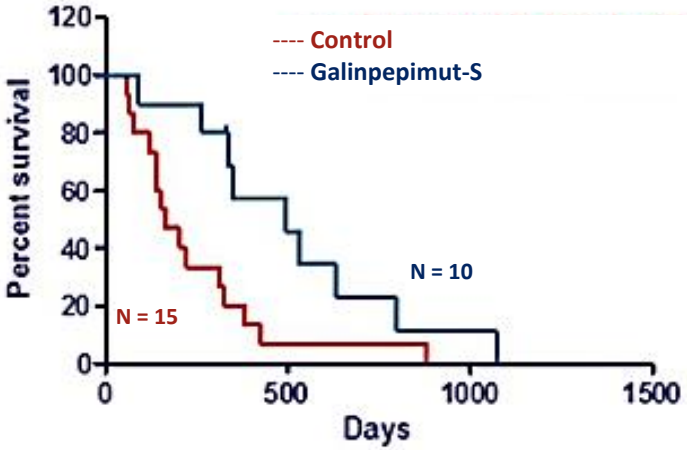
Median Overall Survival (All Age Patients):**GPS** vs **SOC***

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

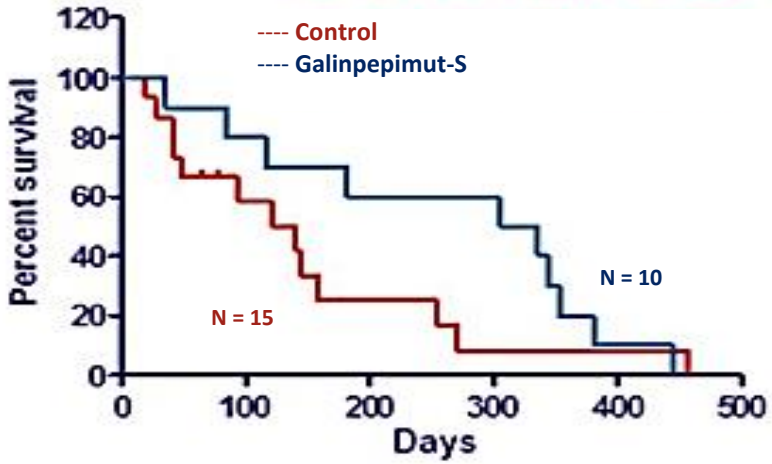
Relapse Free Survival (All Age Patients):**GPS** vs **SOC***

10.5 months vs **4.3 months**

(A) OVERALL SURVIVAL



(B) RELAPSE-FREE SURVIVAL



- Median age→ in GPS arm = 74 years; in control arm = 73 years.
- 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
- % Male patients → 80% in GPS arm; 66.7% in control arm
- 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

* Standard of Care

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

Galinpepimut-S in AML: Safety Analysis

Summary of Treatment-Emergent Adverse Event Analysis in AML CR1

Description	Phase 1 AML CR1 (n=10)[1]	Phase 2 AML CR1 (n=22)[2]
	n (%)	n (%)
Any TEAE	10 (100)	21 (95.5)
Any serious TEAE	0 (0)	1 (4.5)
Any study drug-related TEAE	3 (30)	16 (72.7)
Any study drug-related SAE	0 (0)	1 (4.5)
Any TEAEs leading to discontinuation	0 (0)	1 (4.5)

Drug-Related Adverse Events – Moffitt Trial (n=16*)

Toxicity	Grade 1/2	Grade 3/4
Fatigue	3 (18·75%)	0
Generalized weakness	1 (6·67%)	0
Anorexia	1 (6·67%)	0
Flushing/hot flashes	1 (6·67%)	0
Pain	2 (12·5%)	0
Leukopenia	2 (12·5%)	1 (6·67%)
Anemia	1 (6·67%)	0
Thrombocytopenia	1 (6·67%)	0
Hematoma/abnormal bleeding	2 (12·5%)	0
Nausea	2 (12·5%)	0
Emesis	1 (6·67%)	0
Diarrhea	1 (6·67%)	0
Bloating	1 (6·67%)	0
Transaminitis	1 (6·67%)	0
Hyperbilirubinemia	1 (6·67%)	0
Cough	1 (6·67%)	0
Rash	1 (6·67%)	0
Local injection site reaction	6 (37·5%)	0
Pruritis	1 (6·67%)	0

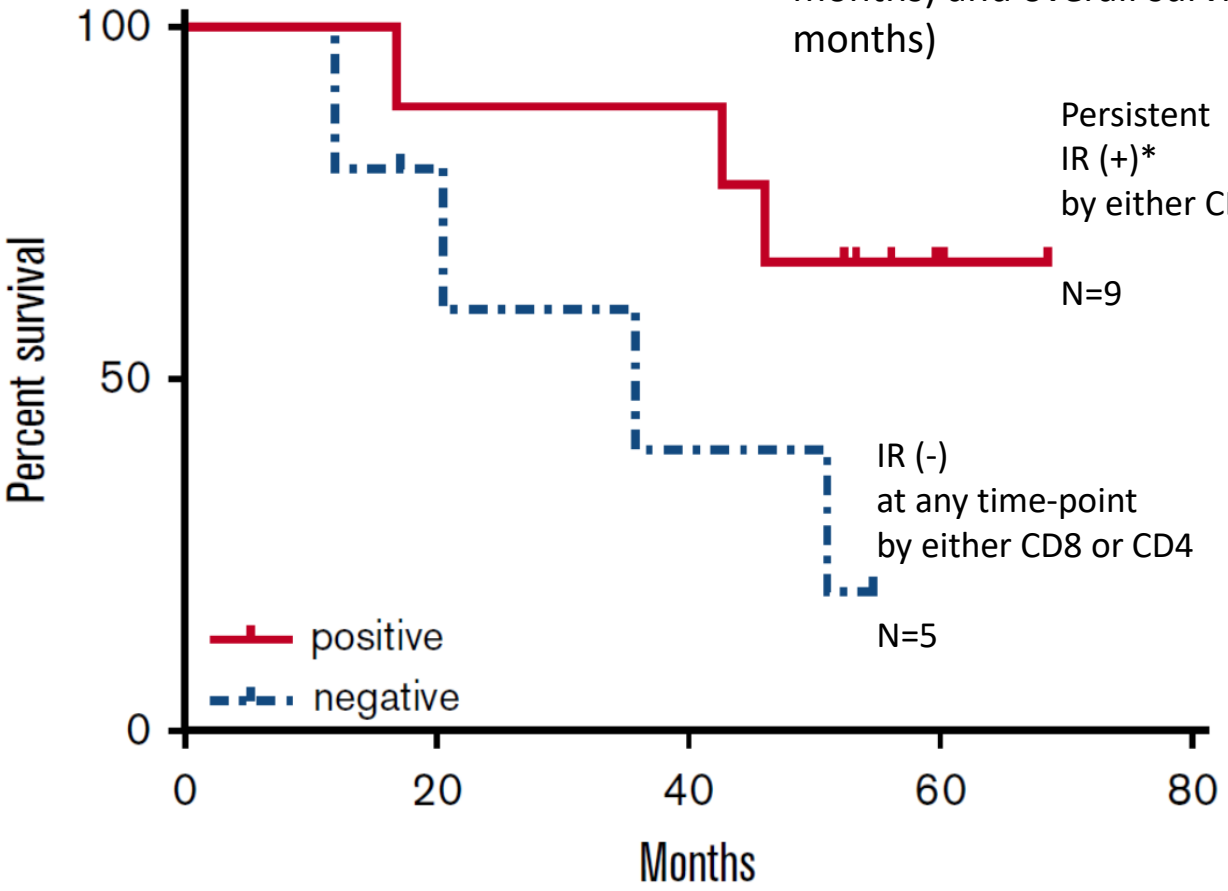
*Safety analysis included 10 pts with AML in CR2 and 6 pts with MD

[1] Maslak PG et al., Blood. 2010 and [2] Maslak PG et al., Blood Adv. 2018

1. Brayer et al., Am J Hematol. 2015

GPS Immunologic Response and Efficacy Correlation

- GPS mechanism of action is based on generation of anti-WT1 T cell clones
- Immunologic response (IR) was closely correlated with both disease-free survival (median N.R. vs. 15.6 months) and overall survival (median N.R. vs. 35.8 months)



Persistent
IR (+)*
by either CD8 or CD4

N=9

IR (-)
at any time-point
by either CD8 or CD4

N=5

mOS
N.R.
35.8 mo

*Persistent antigen-specific IR, mainly CD4+, to any of the 4 peptides in GPS after treatment
CR1: first complete response; IR: immune response; N.R.: not reached; Dx: diagnosis (of AML); mOS: median overall survival

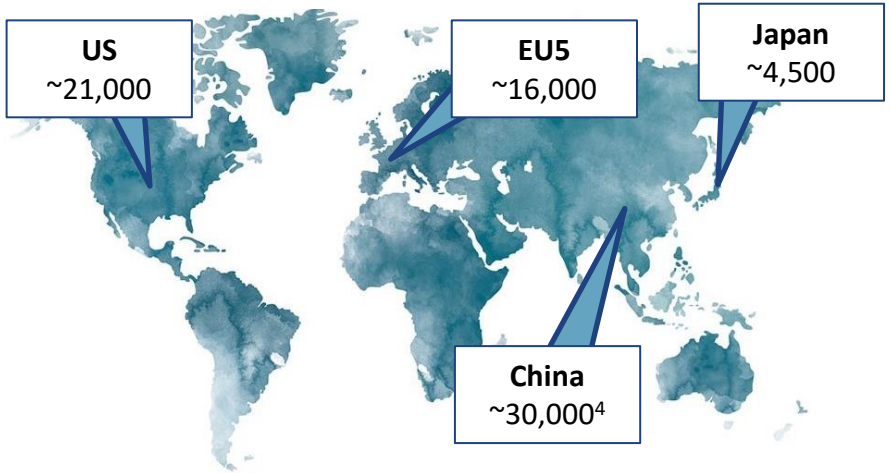
1. Maslak PG, et al. Blood Adv. 2018

Phase 3 Trial Underway in Niche AML CR2 Patient Population within Rare AML Disease – GPS Broad AML Applicability



AML Diagnosis (~77,000 Incident Pts WW)[1]	1st Line Therapy (80% patients)[2]	Complete Remission 1 (CR1) (50-55% patients)[2]	1st Relapse; 2nd Line Therapy (30-35% patients)[2]	Complete Remission 2 (CR2) (12-15% patients)[2]
97% of AML Patients Express WT1	• Chemotherapy • Targeted Therapy • Radiation therapy • Surgery	• CR/MRD[3] determined by biopsy • Consolidation (maintenance) therapy: chemotherapy; targeted therapy continuation; allo/auto SCT • **Phase 1 and 2 data generated in CR1 AML supports GPS potential label expansion in this setting**	• Chemotherapy • Targeted Therapy • Allo SCT • **Post-transplant patients could potentially benefit from GPS treatment**[5]	• CR/MRD as determined by biopsy • Consolidation (maintenance) therapy: chemotherapy, allo-SCT; targeted therapy • **GPS initial label - phase 3 trial ongoing in CR2 AML**

Geographic Incident Adult AML Cases Annually[1]

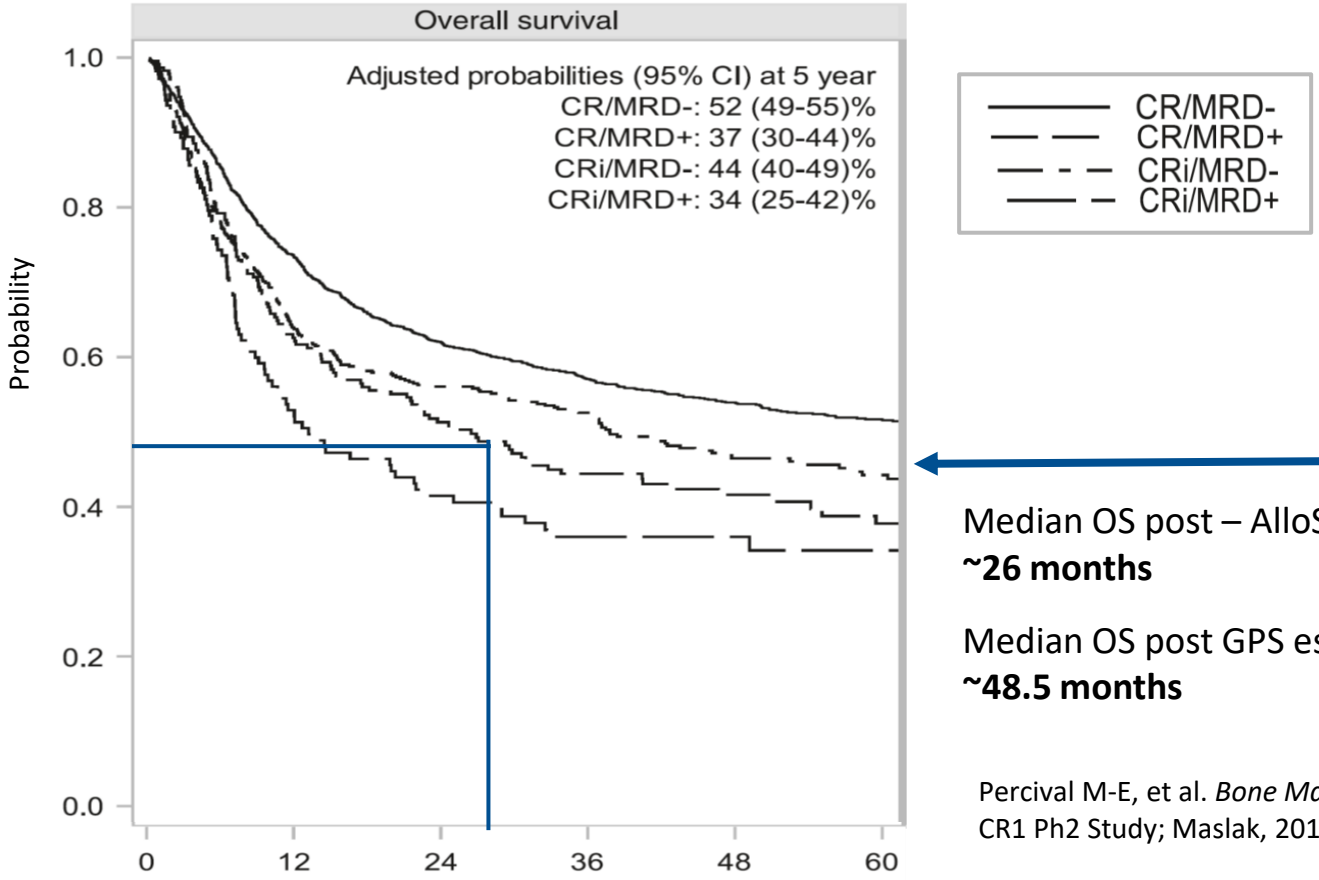


- **GPS** not only has potential for CR2 patients but could be equally important in the CR1 patient population
- Pool of addressable patients further enhanced by fact that CR1 patients who undergo bone marrow transplant (BMT) who are less likely to benefit from it (whom GPS could potentially significantly extend survival) - MRD positive patients – can be identified
- Further potential expansion: patients in complete remission with incomplete peripheral blood recovery (CRi); addition of GPS to any potential maintenance drugs

Notes: 1. Global Data forecasts for 2020 2. Patient segmentation incidence adapted from Kurosowa. S et al. (2010), Haematologica. 95(11): 1857 – 1864; MRD=minimal residual disease; 4. James Moore, Patientworthy.com, July 25, 2019; 5. Percival M-E, et al. *Bone Marrow Transplant*., 2021.

New Data Highlight GPS Opportunities in Bone Marrow Transplant (BMT) Settings

- A comprehensive retrospective analysis published in June 2021 of survival outcomes in 4,280 AML patients showed that BMTs are observed to cure approximately 50% of patients if they have no MRD and approximately 33% of patients with MRD
- Median OS for patients with MRD observed to be approximately one year if they did not have complete peripheral blood recovery after the treatment and approximately two years if they had a complete peripheral blood recovery
- **In Phase 2 CR1 GPS study, patients had complete responses and were MRD positive but not eligible for transplant; median survival was 48.5 months even without transplant, approximately 2x longer than same category of patients who received transplant**



CR1/MRD+

Median OS post – AlloSCT estimate:
~26 months

Median OS post GPS estimate in similar unselected patients:
~48.5 months

Percival M-E, et al. *Bone Marrow Transplant*., 2021
CR1 Ph2 Study; Maslak, 2018.

Design of Phase 3 Registration Enabling Clinical Trial in CR2 AML Patients (REGAL)

ΣELLAS LIFE SCIENCES GROUP

Target Population and Inclusion Criteria

N = 125 to 140
Patients >18 years old

Stratification axes:
- **CR2 vs CRp2 status**
- **Cytogenetics risk category**
- **Duration of CR1 (12 months)**
- **MRD status**

CR2/CRp2 AML Patients post-2L therapy (excluding agents for targeted molecular aberrations)[1]

Ineligible/unable to undergo imminent Allogeneic Stem Cell Transplant (ASCT)

Global Study: approximately 85 clinical sites

R

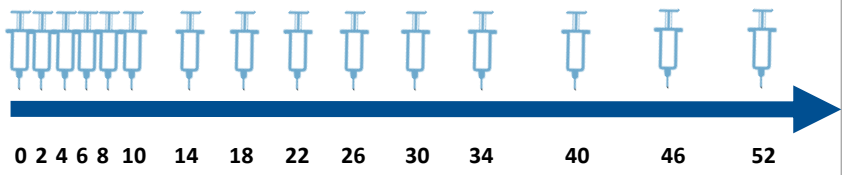
1:1

Treatment: Open Label; Randomised Multi-center Phase 3 Trial

Cohort 1

GPS: 800 µg of peptide mixture; 200 µg of each peptide/dose

Administration Schedule: 1 year



Cohort 2

Best Available Therapy (BAT)
Clinician's choice of 4 regimens (Observation; HMAs and/or Ventoclax and/or Low-dose Ara-C

Endpoints and Interim Analysis

Primary Endpoint: Overall Survival
- At least **90% powered**
- To declare statistical significance: Estimated Hazard Ratio (HR) of 0.636 based on **median OS of 12.6 months (GPS) vs 8.0 months (BAT)**

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



U.S. & Global Lead

Hagop Kantarjian, MD
Distinguished Professor
Head, Dept. of Leukemia
M.D. Anderson Cancer Center
Houston, Texas

Notes: 1. Excluding agents that should be continued in the maintenance setting after achievement of CR2 with usage of 2L regimens containing such agents (e.g. FLT3 inhibitors); CRp2: CR2 with incomplete platelet recovery, i.e., platelet count of ≥60 x 10^9/L (as defined for this study); PB: peripheral blood; BM: bone marrow.



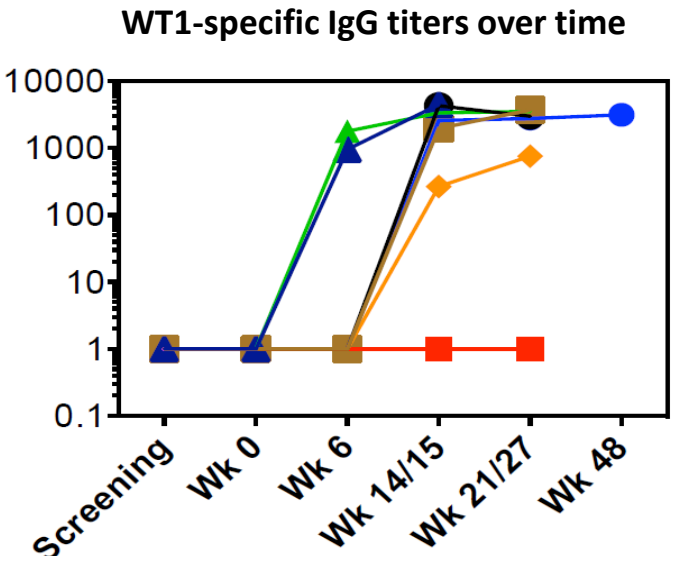
GPS in Combination with Checkpoint Inhibitors

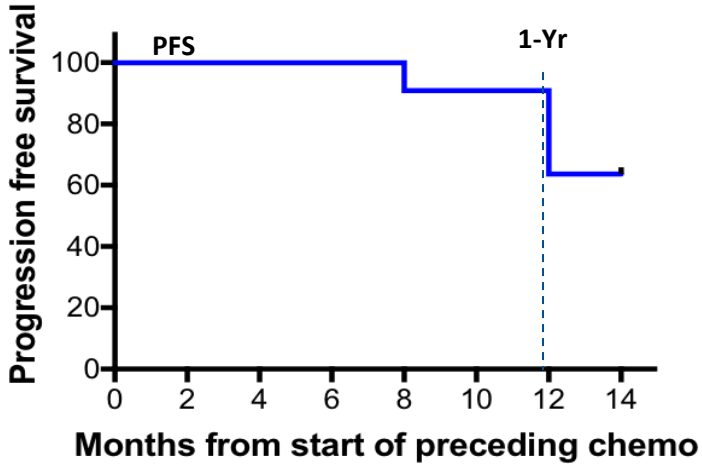
GPS Has Demonstrated Activity and Has Been Well Tolerated in Combo with Checkpoint Inhibitors in WT1 Expressing Solid Tumors

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

Key Points:

1. **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
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)
3. 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]
4. 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).

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

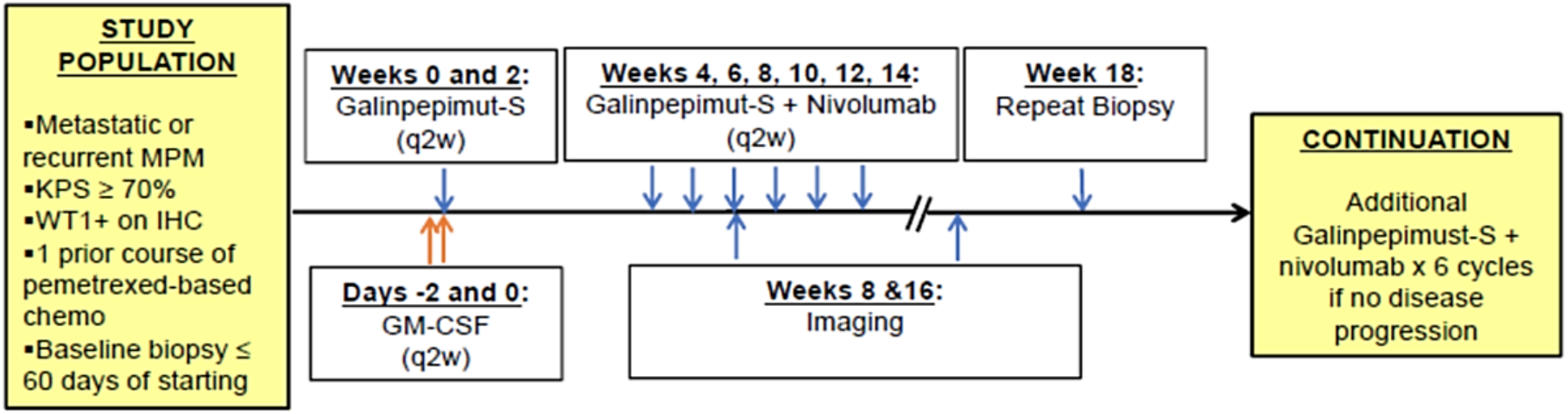
Key Points:

- **Indication:** 2nd or 3rd line WT1(+) relapsed or refractory metastatic ovarian cancer
- **Endpoints**: Safety, immunobiological response, overall response rate (as measured by "response evaluation criteria in solid tumors", or RECIST), progression free survival and overall survival
- **Confirmatory Topline data reported November 2022:**
  - Disease Control Rate (DCR)* of 50.1% for combination at median follow-up of 14.4 months compared to 37.2% in study of single agent checkpoint inhibitor (CI) in similar patient population
  - Median PFS for combination of 12 weeks compared to 8.4 weeks in single agent CI study in similar population
  - Median OS of 18.4 months compared to 13.8 months in single agent CI 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

* *Disease Control Rate=overall response rate + rate of stable disease*

GPS in Combination With Checkpoint Inhibitor: MPM

Open-label Phase 1 Investigator Sponsored Study (IST) of GPS + Nivolumab in relapsed/refractory MPM post 1L SOC Therapy (Pemetrexed) at MSKCC



- Interim data reported June 24, 2021: median overall survival (OS) of 35.4 weeks in patients treated with combination therapy for at least one month (n=4); median OS in R/R patients with standard of care is approximately 28 weeks
- Interim data reported June 8, 2022: 8 evaluable patients with 7 patients receiving combination therapy for at least one month; median OS of 45.7 weeks in 7 patients treated with combination therapy for at least one month; median OS of 40.9 weeks for 8 patients; median OS in R/R patients with standard of care is approximately 28 weeks; median progression free survival was 11.1 for all 8 patients and 11.9 weeks for 7 patients receiving combination therapy
- Safety profile of combination similar to that seen with nivolumab alone
- Completion of enrollment of a target total of 10 evaluable patients expected during the second half of 2022

GPS – Development and Commercialization in Greater China: License Agreement with 3D Medicines



License Agreement with 3D Medicines, Inc.

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

- Sublicensable, royalty-bearing license to develop, manufacture and commercialize GPS and heptavalent GPS (GPS-Plus)
- All therapeutic and diagnostic uses
- Territory: mainland China, Hong Kong, Macau and Taiwan (Greater China)

Financial Terms:

- Upfront cash payment of $7.5 million upon entry of agreement; $3 million in milestones received to date
- Additional potential development, regulatory and commercial milestones totaling up to $193.5 million
- Tiered royalties based upon percentage of annual net sales of licensed products (GPS) in the Greater China territory ranging from high single to low double digits

Current Status:

- 3D Medicines to participate in Phase 3 REGAL study. Participation triggers significant milestone payments to SELLAS, expected to be received in 1H 2023.

GFH009: Next Generation CDK9 Inhibitor



GFH009: Novel Clinical Stage Licensed Small Molecule

GFH009 is a 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 survival of cancer cells, including regulation of short-lived, anti-apoptotic survival proteins (MCL-1 and others) and oncogenes (c-MYC)
- CDK9i can decrease MCL-1 and c-MYC level, which could further induce apoptosis and cell cycle arrest

CDK9 activity is closely 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

CDK9 inhibitors represent an attractive new class of drugs

- First generation CDK9 inhibitors had clinical activity but significant toxicity due to low specificity

Next generation inhibitors have potential for higher specificity and more consistent clinical activity

CDK9 Inhibitors Under Development – Lower CDK9 Specificity

Inhibitor	CDKs	Target cancers	Clinical Trials and Status
Flavopiridol (Alvocidib)	1, 2, 4, 6, 7, 9	Multiple cancers	Numerous; three active trials (NCT03604783; NCT03969420; NCT03593915)
SCH-727965 (Dinaciclib)	1, 2, 5, 9	Multiple cancers	Numerous; four active trials (NCT01676753; NCT03484520; NCT01434316; NCT00937937)
LDC000067	2, 9	Multiple cancers	None (pre-clinical)
SNS-032	2, 7, 9	Adv. solid tumors and adv. B-lymphoid cancers	NCT00446342, NCT00292864 (com)
LY-2857785	7, 9	Multiple cancers	None (pre-clinical)
AT-7519	1, 2, 4, 5, 9	Adv. solid tumors, refractory NHL, MM, MCL, CLL	NCT02503709 (active), NCT00390117, NCT01183949, NCT01652144, NCT01627054 (com)
CYC-202 (Roscovitine)	1, 2, 4, 5, 7, 9	TNBC, NSCLC, adv. solid tumors, Cushing's disease	NCT01333423 (w/d), NCT00372073 (t), NCT00999401,NCT02160730 (t), NCT03774446 (active)
CR-8	1, 2, 4, 5, 7, 9	Neuroblastoma	None (pre-clinical)
CDKI-73 (LS-007)	1, 2, 4 and 9	CLL, AML, ovarian cancer	None (pre-clinical)

CDK9 Inhibitors Under Development – High CDK9 Specificity

Inhibitor	CDKs	Target cancers	Clinical Trials and Status
THAL-SNS-032	9	ALL	None (pre-clinical)
NVP-2	9	ALL	None (pre-clinical)
JSH-150	9	AML and CLL	None (pre-clinical)
BAY-1143572	9	Acute leukemia and adv. cancers	NCT02345382, NCT01938638 (completed/prematurely terminated)
BAY-1251152 (now VIP152)	**9**	**Adv. hem. and adv. solid cancers**	**NCT02745743 (completed), NCT02635672 (active)**
AZD-4573	**9**	**Adv. R/R hem. cancers**	**NCT03263637 (completed), NCT04630756 (active), NCT05140382 (active)**
PRT2527	**9**	**Adv. R/R adv. solid cancers**	**NCT05159518 (active)**
GFH009	**9**	**Adv./R/R hem. cancers**	**NCT04588922 (active)**

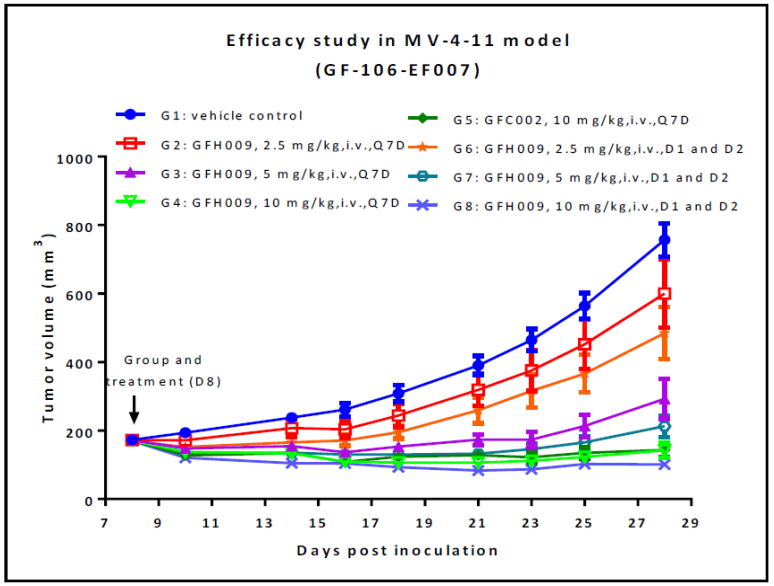
- There are a small number of highly selective CDK9 inhibitors in active clinical development in the US
  - BAY-1143572 (Atuveciclib) development was discontinued due to toxicity in P1
- BAY-1251152 (now VIP152): Two Phase 1 trials completed and one Phase 1 trial ongoing
  - Phase 1 monotherapy r/r NHL and r/r solid cancers; Phase 1 monotherapy in r/r AML; Phase 1 monotherapy in DHL (double hit lymphoma)
- AZD-4573: Phase 1 completed, 2 ongoing trials:
  - Phase 2 monotherapy in r/r PTCL and r/r cHL; Phase 1/2 combination with acalabrutinib (2nd gen BTK inhibitor)
- PRT2527: Phase 1 ongoing, started in Jan 2022
  - Phase 1 monotherapy: Adult sarcomas of any histology, CRPC, ER+ BC, TNBC, and solid tumors with known MYC ampl.
- GFH009: Phase 1 trial ongoing in China and United States
  - Phase 1 monotherapy trial in r/r AML, CLL/SLL and lymphoma

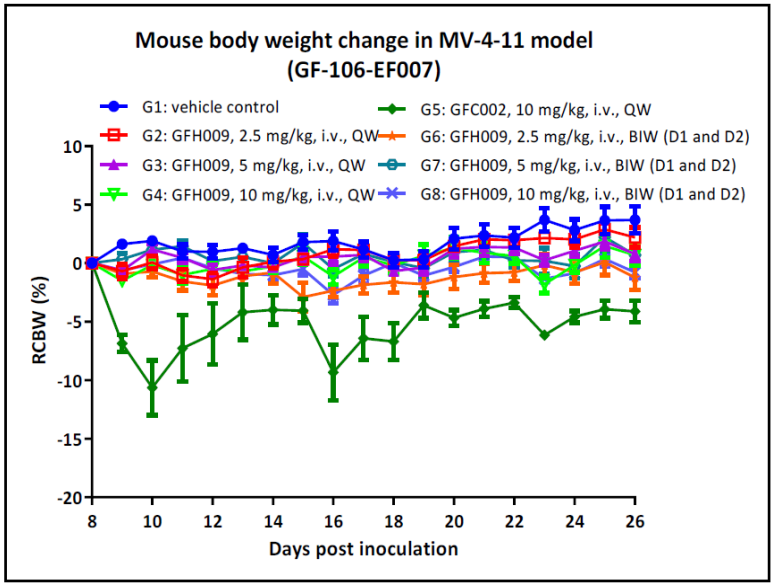
*VIP152 is a new name for BAY-1251152 after in-licensing from Bayer by Vincerx Pharma in October 2020

Clinical Stage Selective CDK9s Under Development: Preclinical Comparison

Preclinical data shows highly specific GFH009 CDK9 affinity and potential for superior safety and efficacy compared to BAY-125112

Cell lines	GFH009 IC50 (72h)	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





GFC002 code for BAY-1251152

Design of Ongoing GFH009 Phase 1 Trial

ΣELLAS LIFE SCIENCES GROUP

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 P 1

Two part study:

In the dose escalation part, the dose levels escalated following the Bayesian optimal interval (BOIN) design.

In the expansion part, patients will be assigned based on tumor type(s).

Administration and Dose Escalation Algorithm

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

Planned dose levels completed:	Additional dose levels added:
	GFH009 60 mg 1D/7D
	GFH009 45 mg 1D/7D
GFH009 30 mg 2D/7D	GFH009 30 mg 1D/7D
GFH009 22.5mg 2D/7D	
GFH009 15 mg 2D/7D	
GFH009 9 mg 2D/7D	
GFH009 4.5 mg 2D/7D	
GFH009 2.5 mg 2D/7D	

Endpoints and Assessments

Primary Endpoints:
Safety and Tolerability of GFH009:
Dose Limiting Toxicities (DLTs)
Time Frame: 21 days (The incidence of DLTs)
Safety and Tolerability of GFH009: 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

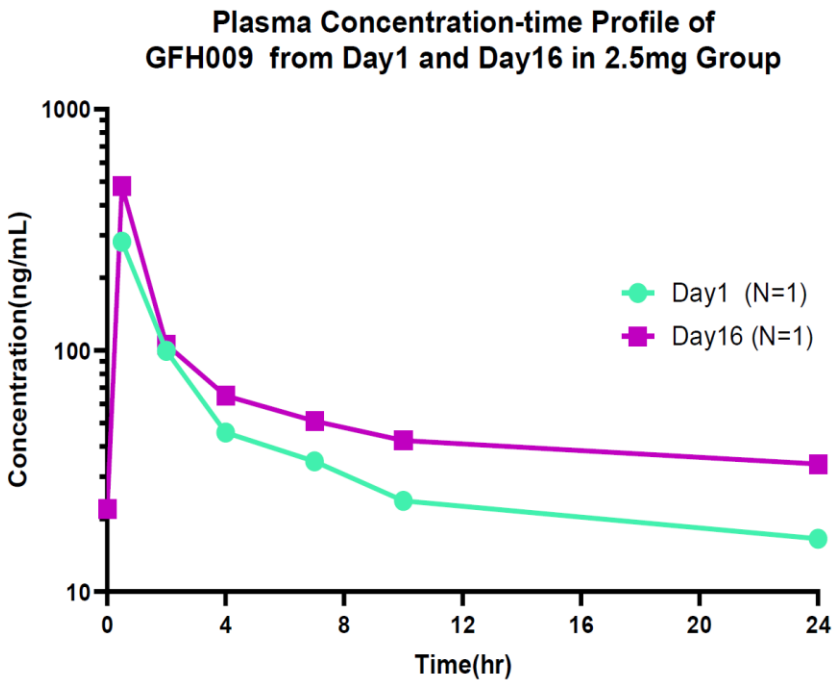
PK parameter AUC0-∞ (Area under the plasma concentration-time curve (from zero to infinity)
Time Frame: approximately 3 months

Pharmacodynamic Assessments:
Mcl 1 levels
C-Myc levels
(% change during treatment)

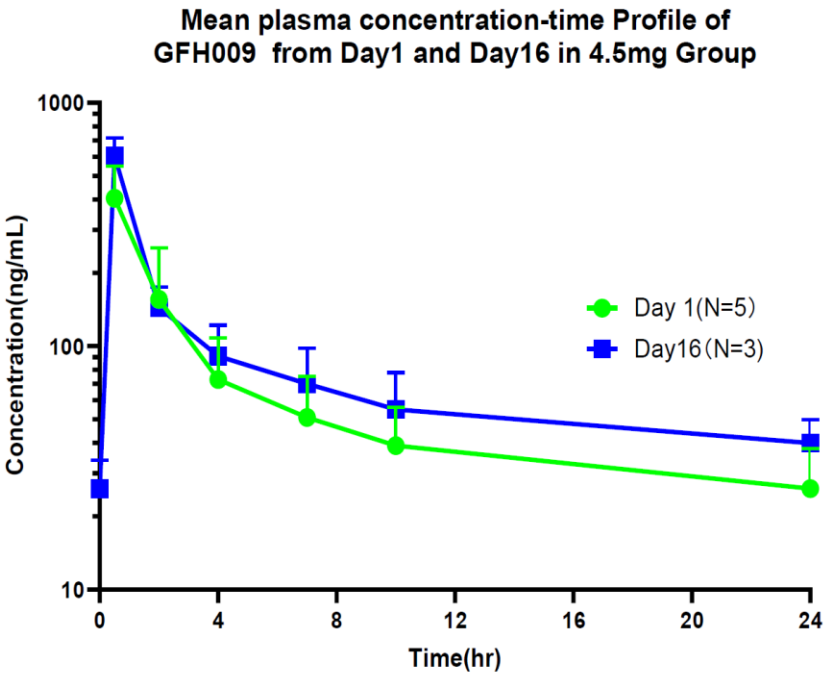
Favorable Early PK Data in the Ongoing Phase 1 Clinical Trial

- At the initial dose levels of 2.5mg and 4.5mg, rapid clearance from plasma was observed which is consistent with low expected toxicity [1]

Cohort 1: 2.5mg



Cohort 2: 4.5mg



- For VIP152 the mean elimination half-life was 3-9 hours [2]

(1) Company data
(2) M Byrne et al, Blood (2018) 132 (Supplement 1)

Select Clinical Stage CDK9 Inhibitors: Monotherapy Clinical Results

Inhibitor	Patient population	Results
AZD-4573	Adv. R/R hem. cancers 44 pts treated : 22 each in Arms A (lymphoma) and B (AML)	All pts had discontinued treatment, mainly due to disease progression (47.7%) or AEs (22.7%). SAEs in 88.6%. Treatment-related AEs (TRAEs) occurred in 93.2% of pts and were serious in 59.1%, but none led to death. 18 mg dose not tolerated in either arm. RP2D was 12 mg QW in Arm A and 9 mg QW in Arm B. In Arm A, there was 1 complete and 1 partial response in 17 DLBCL pts[1]
PRT2527	Adv. R/R solid cancers	NA
BAY-1251152 (now VIP152)	Refractory AML 21 pts	G3/4 AEs in >10% pts: neutropenia, anemia, leukocytosis and lung infection; no objective responses, dose dependent short lived blast decreases[2]
BAY-1251152 (now VIP152)	R/R NHL and R/R solid ca. 31 pts	No DLTs at DLs 5, 10 and 15 mg; 1 DLT at 22.5 mg (G4 neutropenia) MTD 30 mg, 3/9 pts with G3/4 neutropenia; no responses; 9/31 pts (29%) had SD with tumor burden reductions up to 40%. Durable SD in DH-DLBCL, PAC and SGC[3]
BAY-1251152 (now VIP152)	R/R DH-DLBCL 7 pts	30 mg DL; G3/4 AEs: fatigue, lymphocytopenia, neutropenia (each 1/7 pts, 14%) and tumor pain (2/7 pts, 29%); 2/7 (29%) metabolic CRs[4]
GFH009	Adv.R/R hem. cancers Enrolled 53 pts to date	No DLTs, MTD not reached, 24 SAEs in 18 subjects (32%), 6 (8%) potentially drug-related (respiratory infections) No drug-related deaths. One (2%) pt discontinued due to AE; one (2%) pt had dose reduction due to AE. Lymphoma pts: 2 pts had a partial response and 5 pts had stable disease. AML pts: 2 pts had blast count deceased of 50% in BM. Two pts on treatment for more than 10 weeks and still ongoing[5]

(1) Safety, Tolerability, Pharmacokinetics (PK) and Preliminary Antitumor Activity of the Cyclin-Dependent Kinase-9 (CDK9)Inhibitor AZD4573 in Relapsed/Refractory Hematological Malignancies: A Phase 1 First-in-Human Study, ASH 2022; (2) M Byrne et al, Blood (2018) 132 (Supplement 1): 4055; (3) J Diamond et al Journal of Clinical Oncology (2018) Volume 36, Issue 15_suppl; (4) V Moreno et al, Journal of Clinical Oncology (2021) Volume 39, Issue 15_suppl; (5) Company data

GFH009 Preclinical and Clinical Data Differentiation

GFH009 compares favorably based on preclinical and clinical data:

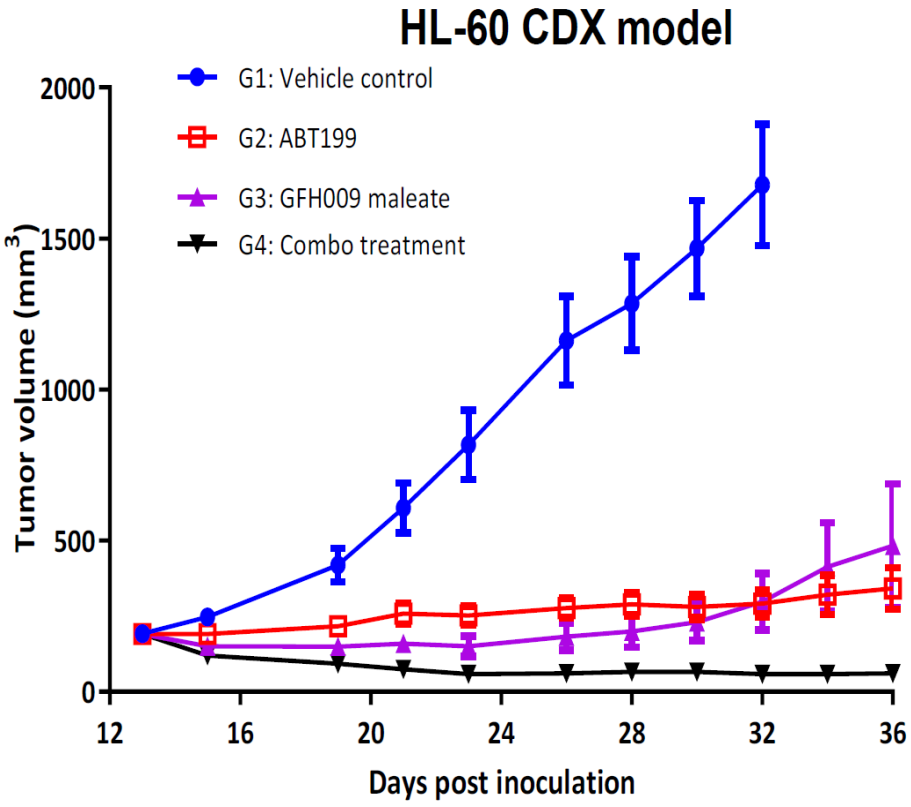
- **Better in vitro anti-proliferative activity** in a preclinical model directly comparing GFH009 vs. BAY1251152 (GFC002)
- **Tumor reduction burden at very low dose levels** compared to clinical results in separate trials of competitors:
  - Partial responses and stable disease as monotherapy in relapsed/refractory lymphoma patients after several lines of therapy starting at 4.5 mg DL.
  - Significant anti-leukemic effect at 9mg and 15mg dose levels given twice a week in AML patients resistant to the multiple standard-of-care treatments, with two patients refractory to, or relapsed after, venetoclax treatment with ≥50 % decrease in bone marrow blasts following GFH009 monotherapy.

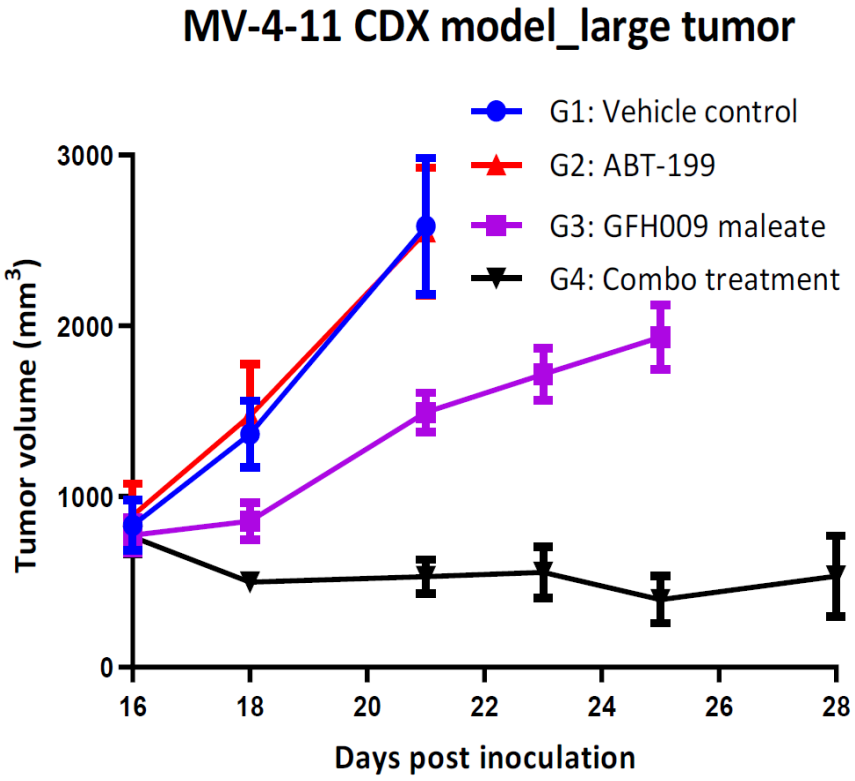
Potentially improved safety profile:

- Superior safety to BAY-1251152 (VIP 152) in direct preclinical mouse model comparison
  - Similar efficacy with significantly better safety at the same dose level
    - At same dose and administration schedule, comparable/slightly better tumor growth inhibition with significantly less mouse weight loss
    - Further improvement of efficacy at more frequent dosing of GFH009 with no deterioration of safety
- No dose-limiting toxicities, including no grade 3/4 neutropenia DLTs, at all studied dose levels including 30 mg twice-weekly and 3 pts at 45 mg once weekly.

GFH009 Synergy with Venetoclax

In preclinical models, GFH009 was able to convert cancer cells partially or completely resistant to venetoclax (ABT-199) to being highly sensitive to the combination





GFH009 Clinical Development Plan Following Completion of Phase 1 Clinical Trial

Proposed Plan for United States (SELLAS)

- AML: Phase 2 trial combination with venetoclax/azacitidine
  - Venetoclax/azacitidine patients refractory to 2-4 cycles of treatment
  - Venetoclax/azacitidine patients relapsed on continued venetoclax/azacitidine
  - Primary endpoint complete response rate
- Phase 2 trial in solid tumors
- Potential development program for pediatric cancers
  - Possibility of obtaining a rare pediatric disease priority review voucher

China (Genfleet)

- TPLL and NK/T lymphoma in 2nd line or above → Phase 2: Monotherapy and potential pivotal study as a single arm
- Double hit lymphoma in 2nd line or above → Phase 2: Monotherapy and potential pivotal study as a single arm
- DLBCL (ABC) in 2nd line or later → Phase 2: Monotherapy
- DLBCL (ABC) in 1st line → Phase 2: In combination with BTK inhibitor or R-CHOP
- AML → Phase 2: Combined with venetoclax or azacitidine
- Hodgkin's' lymphoma 2nd line or above post anti-PD-1 antibody treatment → Phase 2: Monotherapy and potential pivotal study as a single arm

Key Takeaways

✓ Two novel clinical stage cancer therapies with minimal competition

- GPS: a sophisticated, highly differentiated immunotherapeutic
  - Good tolerability, consistent and notable signals of clinical benefit
  - Heteroclitic peptides increase immune response
  - Production of both CD4 and CD8 WT1-specific activated cells
- GFH009
  - Highly selective next generation CDK9 inhibitor
  - Broad applicability across different cancer types
  - Significant potential for combination with hematological cancer standard of care

✓ Near-term milestones

- GPS:
  - Phase 3 REGAL trial for AML CR2 patients
    - Interim data expected late 2023 or early 2024
  - Phase 1/2 basket clinical trial in combination with checkpoint inhibitor in advanced ovarian cancer
    - Final topline data reported November 2022
  - Phase 1 clinical trial in MPM in combination with checkpoint inhibitor
    - Additional interim data reported June 2022; completion of enrollment expected by end of 2022
- GFH009:
  - Phase 1 study expected completion end of 2022
  - Phase 2 studies expected commencement 1Q 2023

SELLAS LIFE SCIENCES GROUP

Thank You!

For additional information, please contact:

SELLAS Life Sciences Group, Inc. (Nasdaq: SLS)
646.200.5278
info@sellaslife.com
SELLAS@kcsa.com