NEWS RELEASE

SELLAS Life Sciences Presents Preclinical Data Demonstrating SLS009 Activity in Pancreatic Cancer Models at the 2026 AACR Conference on Pancreatic Cancer

2026-09-25

- SLS009 increased apoptosis more than six-fold versus daraxonrasib in a MYC-amplified, daraxonrasib-resistant patient-derived pancreatic cancer organoid model -

- Combination of SLS009 and daraxonrasib further increased apoptosis to 36.5% and necrosis to 30.6%, supporting evaluation of CDK9 inhibition as a potential strategy to enhance RAS-directed therapy -

- SLS009 combined with BET inhibition demonstrated synergistic activity and sustained suppression of MYC, providing additional evidence of SLS009 activity against MYC-driven pancreatic cancer biology -

NEW YORK, Sept. 25, 2026 (GLOBE NEWSWIRE) -- SELLAS Life Sciences Group, Inc. (NASDAQ: SLS) (“SELLAS” or the “Company”), a late-stage clinical biopharmaceutical company focused on the development of novel therapies for a broad range of cancer indications, today announced preclinical data from studies evaluating SLS009 (tambiciclib), its highly selective cyclin-dependent kinases 9 (CDK9) inhibitor, in patient-derived organoid models of pancreatic ductal adenocarcinoma (PDAC). The data are being presented at the American Association for Cancer Research (AACR) Conference on Pancreatic Cancer: New Frontiers in Biology and Therapeutic Development, being held September 25–28, 2026, in San Diego. The timing of this announcement reflects AACR’s embargo policy, under which the data presented at the conference were restricted from publication until 1:00 p.m. ET today.

The studies, conducted in collaboration with researchers at the University of Wisconsin–Madison, evaluated SLS009 in MYC-amplified PDAC models, including a model resistant to the recently approved RAS inhibitor daraxonrasib

(RMC-6236), as well as in combination with the BET inhibitor ZEN3694. MYC amplification is a biologically recognized mechanism of RAS targeting resistance.

“These findings provide encouraging preclinical evidence that CDK9 inhibition may enhance the activity of RAS-directed therapy in pancreatic cancer, including in the setting of MYC-associated resistance,” said Dragan Cicic, MD, Senior Vice President, Clinical Development of SELLAS. “In a daraxonrasib-resistant patient-derived model, SLS009 demonstrated substantially greater activity than daraxonrasib alone and further increased apoptosis and necrosis when the two agents were combined. Together with the synergistic activity observed with BET inhibition, these data support a broader strategy of using SLS009 to disrupt transcriptional programs that may contribute to resistance to RAS-targeted therapies and provide a strong rationale for further evaluation in pancreatic cancer patients.”

In a MYC-amplified, daraxonrasib-resistant patient-derived PDAC organoid model, SLS009 at 200 nM, daraxonrasib at 100 nM, and the combination of both agents were evaluated. Daraxonrasib was administered continuously, while SLS009 was removed after 24 hours to approximate its in vivo pharmacokinetic profile, with apoptosis and necrosis assessed at 72 hours.

A separate MYC-amplified patient-derived PDAC organoid model evaluated SLS009 in combination with the BET inhibitor ZEN3694. The combination demonstrated synergistic activity, including increased cancer cell death and sustained suppression of MYC transcription. Notably, these effects were observed at a ZEN3694 concentration substantially below reported physiologically achievable exposure levels.

Key findings:

- 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%.
- The combination of SLS009 and daraxonrasib further increased cancer cell death, inducing 36.5% apoptosis and 30.6% necrosis, compared with 17.9% and 14.5%, respectively, with SLS009 alone and 2.8% and 2.7%, respectively, with daraxonrasib monotherapy.
- SLS009 combined with ZEN3694 demonstrated synergistic activity, producing greater apoptosis and necrosis than either agent alone.
- The SLS009/ZEN3694 combination produced sustained suppression of MYC RNA and reduced expression of MYC and MCL-1 proteins, consistent with disruption of transcriptional pathways supporting tumor cell survival.

Together, the findings support further investigation of CDK9 inhibition as a strategy to enhance RAS-directed

therapy and potentially address MYC-associated resistance in pancreatic cancer. The BET combination data provide additional mechanistic support for SLS009-based approaches designed to disrupt MYC-dependent transcriptional programs and suggest the potential to enhance BET inhibition at lower drug exposures.

“MYC is a particularly challenging oncogenic driver because it has historically been difficult to target directly,” said Jeremy D. Kratz, MD, Assistant Professor of Medicine and Principal Investigator at the University of Wisconsin–Madison. “Across these studies, CDK9 inhibition produced substantial activity in MYC-amplified pancreatic cancer models through two distinct therapeutic strategies. The activity of SLS009 supports its activity in a model with de novo daraxonrasib-resistance and together with the synergistic transcriptional suppression observed with BET inhibition, provides a strong rationale for further investigation of SLS009-based combinations in molecularly defined subsets of pancreatic cancer.”

Poster presentation details:

Title: Elucidating MYC allelic imbalance and therapeutic response in pancreatic ductal adenocarcinoma via patient-derived organoids
Authors: Sawyer AG, Flannagan LE, Esguerra PN, Hossan MS, Kratz JD
Poster Number: A036 – September 27, 2026: 5-7pm PST

Title: Synthetic Lethality Through Combined BET and CDK9 Inhibition in MYC-Amplified Pancreatic Ductal Adenocarcinoma
Authors: Esguerra PN, Cadarso M, Livingwell S, Hossan MD, Wong O, Kratz JD
Poster Number: B127 – September 27, 2026: 5-7pm PST

Title: Targeting dual CDK9 and KRASG12D selective inhibition as a novel combination therapy in pancreatic ductal adenocarcinoma
Authors: Cadarso M, Esguerra P, Flannagan L, Hossan MS, Kratz JD
Poster Number: B043 – September 27, 2026: 5-7pm PST

The posters will be available on SELLAS’ website following the conference.

About SELLAS Life Sciences Group, Inc.

SELLAS is a late-stage clinical biopharmaceutical company focused on the development of novel therapeutics for a broad range of cancer indications. SELLAS’ lead product candidate, GPS, is licensed from Memorial Sloan Kettering Cancer Center and targets the WT1 protein, which is present in an array of tumor types. GPS has the potential as a monotherapy and combination with other therapies to address a broad spectrum of hematologic malignancies and

solid tumor indications. The Company is also developing SLS009 (tambiciclib) – potentially the first and best-in-class differentiated small molecule CDK9 inhibitor with reduced toxicity and increased potency compared to other CDK9 inhibitors. Data suggests that SLS009 demonstrated a high response rate in AML patients with unfavorable prognostic factors including ASXL1 mutation, commonly associated with poor prognosis in various myeloid diseases. For more information on SELLAS, please visit **www.sellaslifesciences.com**.

Forward-Looking Statements

This press release contains forward-looking statements. All statements other than statements of historical facts are "forward-looking statements," including those relating to future events. In some cases, forward-looking statements can be identified by terminology such as "plan," "expect," "anticipate," "may," "might," "will," "should," "project," "believe," "estimate," "predict," "potential," "intend," or "continue" and other words or terms of similar meaning. These statements include, without limitation, statements related to the GPS clinical development program, including the REGAL study and the timing of future milestones related thereto. 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 with oncology product development and clinical success thereof, the uncertainty of regulatory approval, and other risks and uncertainties affecting SELLAS and its development programs as set forth under the caption "Risk Factors" in SELLAS' Annual Report on Form 10-K filed on March 19, 2026 and in its other SEC filings. Other risks and uncertainties of which SELLAS is not currently aware may also affect SELLAS' forward-looking statements and may cause actual results and the timing of events to differ materially from those anticipated. 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.

Investor Contact

John Fraunces
Managing Director
LifeSci Advisors, LLC
jfraunces@lifesciadvisors.com

Media Contact

Joan Bosisio

VP, Scientific & Medical Communications
Simpson Healthcare
jbosisio@simpsonhealthcare.com

Source: SELLAS Life Sciences Group, Inc.