Sumitomo Pharma

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Sumitomo Pharma America to Present Preliminary Clinical Data Evaluating Investigational Oncology Agents TP-3654 and DSP-5336 at the American Society of Hematology Annual Meeting

– ASH Preliminary Data Includes New Clinical Results in Relapsed or Refractory Myelofibrosis (TP-3654) and Relapsed or Refractory Acute Leukemia (DSP-5336) –

CAMBRIDGE, Mass., Nov. 3, 2023 – Sumitomo Pharma America, Inc. (SMPA) today announced preliminary clinical data for investigational agents TP-3654, a selective oral PIM1 kinase inhibitor, and DSP-5336, an inhibitor of the menin and mixed-lineage leukemia (MLL) protein interaction. These data will be presented at the 65th American Society of Hematology (ASH) Annual Meeting & Exposition, held December 9-12 in San Diego, Calif.

Preliminary results from the ongoing Phase 1/2 study of TP-3654 monotherapy in patients with relapsed or refractory myelofibrosis who were previously treated with or ineligible for a JAK inhibitor will be presented in an oral presentation at ASH. In this study, oral TP-3654 was well-tolerated with limited myelosuppressive adverse events. TP-3654 exhibited early signs of clinical activity including spleen volume reduction, total symptom score improvement, and correlating cytokine reductions.

SMPA will also present a poster on preliminary clinical data from the ongoing Phase 1/2 first-in-human study of oral DSP-5336, in patients with relapsed or refractory acute leukemia. Preliminary data showed that DSP-5336 was well-tolerated with no dose limiting toxicities, including no observed cardiac signals. Target pharmacodynamic changes were observed with treatment, including rapid decreases in genes commonly expressed in leukemia (HOXA9, MEIS1, and PBX3). These changes were seen particularly in patients with acute myeloid leukemia characterized by a KMT2A (MLL) gene rearrangement or a mutation in the NPM1 gene.

“We are encouraged by the early signs of clinical activity shown in the preliminary results from the TP-3654 and DSP-5336 Phase 1/2 trials. We look forward to sharing the data and having important scientific engagement involving both agents at the annual ASH meeting in December,” said Jatin Shah, M.D., Chief Oncology Development Officer, SMPA. “Improving patient outcomes and developing new oncological treatments is a primary focus for SMPA and we remain committed to exploring the potential of our diverse research pipeline.”

Abstract Title	Detail	Lead Author
Phase 1/2 Study of TP-3654, a Selective PIM1 Kinase Inhibitor: Preliminary Data	Session Name: 634. Myeloproliferative Syndromes: Clinical and	Lindsay A.M. Rein, M.D.

Showed Clinical Activity and Cytokine Reductions in Relapsed/Refractory Myelofibrosis Patients	Epidemiological: Myelofibrosis: New Therapeutic Frontiers Session Date: Sunday, December 10 Session Time: 4:30 p.m. - 6:00 p.m. PST Presentation Time: 4:45 p.m. PST Location: Marriott Marquis San Diego Marina, Pacific Ballroom Salons 21-22 Oral Podium Presentation	
Phase 1/2 First-in-Human Study of the Menin-MLL Inhibitor DSP-5336 in Patients with Relapsed or Refractory Acute Leukemia	Session Name: 616. Acute Myeloid Leukemias: Investigational Therapies, Excluding Transplantation and Cellular Immunotherapies: Poster II Session Date: Sunday, December 10 Presentation Time: 6:00 p.m. - 8:00 p.m. PST Location: San Diego Convention Center, Halls G-H Poster Presentation	Naval Daver, M.D.

About TP-3654

TP-3654 is an oral investigational inhibitor of PIM1 kinase, which has shown potential antitumor and antifibrotic activity through multiple pathways, including induction of apoptosis in preclinical models.[1,2] TP-3654 was observed to inhibit proliferation and increase apoptosis in murine and human hematopoietic cells expressing the clinically relevant JAK2V617F mutation.[2] TP-3654 alone and in combination with ruxolitinib showed white blood cell and neutrophil count normalization, and also reduced spleen size and bone marrow fibrosis in JAK2V617F and MPLW515L murine models of myelofibrosis.[2] The safety and efficacy of TP-3654 is currently being clinically evaluated in a Phase 1/2 study in patients with intermediate and high-risk myelofibrosis (NCT04176198). The U.S. Food and Drug Administration (FDA) granted Orphan Drug Designation for TP-3654 for the indication of myelofibrosis in May 2022.

About DSP-5336

DSP-5336 is an investigational small molecule inhibitor of the menin and mixed-lineage leukemia (MLL) protein interaction. Menin is a scaffold nuclear protein that plays various key roles in biological pathways, including cell growth regulation, cell cycle control, genomic stability, bone development, and hematopoiesis.[3,4] In preclinical studies, DSP-5336 has shown selective growth inhibition in human acute leukemia cell lines with KMT2A (MLL) rearrangements or NPM1 mutations.[3,5] The safety and efficacy of DSP-5336 is currently being clinically evaluated in a Phase 1/2 dose escalation/dose expansion study in patients with relapsed or refractory acute leukemia (NCT04988555). The FDA granted Orphan Drug Designation for DSP-5336 for the indication of acute myeloid leukemia in June 2022.

About Sumitomo Pharma
Sumitomo Pharma Co., Ltd. is a global pharmaceutical company based in Japan with key operations in the U.S. (Sumitomo Pharma America, Inc.), Canada (Sumitomo Pharma Canada, Inc.), and Europe (Sumitomo Pharma Switzerland GmbH) focused on addressing patient needs in psychiatry & neurology, oncology, urology, women's health, rare disease, and cell & gene therapies. With several marketed products in the U.S., Canada, and Europe, a diverse pipeline of early- to late-stage assets, and in-house advanced technology capabilities, we aim to accelerate discovery, research, and development to bring novel therapies to patients sooner. For more information, please visit https://www.us.sumitomo-pharma.com and LinkedIn to follow us.

For a copy of this release, visit Sumitomo Pharma America's website at https://www.us.sumitomo-pharma.com

References

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4. Matkar S, Thiel A, Hua X. Menin: a scaffold protein that controls gene expression and cell signaling. Trends Biochem Sci. 2013; 38(8):394-402. doi:10.1016/j.tibs.2013.05.005.
5. Kuhn MWM, Song E, Feng Z, et al. Targeting chromatin regulators inhibits leukemogenic gene expression in NPM1 mutant leukemia. Cancer Discov. 2016; 6(10):1166-1181. doi:10.1158/2159-8290.CD-16-0237.