Alexo Therapeutics Presents Preclinical Data Demonstrating that ALX148 Bridges Innate and Adaptive Immunity to Enhance Antitumor Immune Response at the 59th Annual ASH Meeting

DUBLIN, Ireland and SOUTH SAN FRANCISCO, Calif. – December 9, 2017 – Alexo Therapeutics, a clinical-stage immuno-oncology company developing therapies to block the CD47 checkpoint mechanism, today presented preclinical data for its lead candidate, ALX148. Data from the oral presentation at the 59th American Society of Hematology Annual Meeting (ASH) held in Atlanta, Georgia demonstrate that ALX148 triggers a broad antitumor response by bridging innate and adaptive immunity.

"CD47 blockade is among the most exciting new immuno-oncology approaches because of its potential to combine with a broad range of anti-cancer therapies," said Hong Wan, Ph.D., Chief Scientific Officer of Alexo. "We are excited by our results with ALX148, which is differentiated from other CD47 blockers by its unique design. ALX148 triggers a broad antitumor immune response by both innate and adaptive immune systems and enhances the efficacy of checkpoint inhibitors and targeted anti-cancer antibodies in preclinical models. Given the favorable single agent safety profile observed in the clinic, we look forward to exploring ALX148 combinations with anti-cancer therapeutics in our clinical program."

Tumor cells over-express the CD47 protein, which binds its partner SIRPα on dendritic cells and other myeloid cells, leading to suppression of immune function. ALX148 was shown to bind the CD47 protein with high affinity and prevent its interaction with SIRPα. To see if blocking this interaction with ALX148 could activate an antitumor immune response, Alexo scientists used tumor-bearing mice with normal immune systems.

In these syngeneic tumor studies, ALX148 enhanced the antitumor activities of immune checkpoint inhibitors targeting PD-1 and PD-L1, leading to decreased tumor size and increased survival. ALX148 induced a broad antitumor immune response, promoting dendritic cell activation and decreasing the suppressive functions of tumor associated macrophages, leading to activation of tumor-specific responses by cytotoxic T cells. These results show that ALX148 bridges innate and adaptive immunity to activate multiple immune cell types against tumors and provide a rationale for combination with other immunotherapeutic approaches.

ALX148 is designed to avoid the targeting of normal CD47-expressing cells, thus minimizing treatment-related toxicities. In non-human primate toxicology studies, ALX148 was well tolerated with no adverse effects on hematological parameters. Preliminary clinical data presented at the 32nd Annual Meeting of the Society for

Immunotherapy of Cancer (SITC) showed that ALX148 as a single agent was generally well tolerated in patients with advanced malignancy, with no dose-dependent impact on normal blood cells.

About ALX148

ALX148 is a fusion protein comprised of an engineered high affinity CD47 binding domain of SIRPα linked to an inactive Fc region of human immunoglobulin. ALX148 potently and specifically binds CD47 and blocks its interaction with SIRPα, thus inhibiting a key immune checkpoint mechanism exploited by cancer cells. ALX148 is currently being investigated in a first-in-patient, phase 1 study in patients with advanced solid tumors and lymphoma (NCT03013218). Upon completion of the single agent portion, the second half of the trial will evaluate ALX148 in combination with checkpoint inhibitors and targeted anti-cancer antibodies.

About Alexo Therapeutics

Alexo Therapeutics is a clinical-stage immuno-oncology company developing therapies that block the CD47 checkpoint mechanism, which is exploited by cancer cells to evade the immune system. Our lead candidate, ALX148, is a fusion protein comprised of an engineered high affinity CD47 binding domain of SIRPα linked to an inactive Fc region of human immunoglobulin. ALX148 is designed to enhance the efficacy of antibody-based therapies and is in clinical development for a broad range of tumor types.
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