METHODS OF INCREASING CELL PHAGOCYTOSIS

CROSS-REFERENCE TO RELATED APPLICATIONS

This application claims the benefit of priority based on U.S. Provisional Application No. 63/245,540, filed September 17, 2021, the content and disclosure of which is incorporated herein by reference in its entirety.

BACKGROUND

CD47 is a protein expressed on normal cells serving as an immune checkpoint to inhibit macrophage-mediated destruction of its host cells. It binds to signal regulatory protein α (SIRPα), a membrane glycoprotein expressed on myeloid cells. The CD47-SIRPα binding forms a signaling axis marking the host cell as self to avoid phagocytosis by the immune system.

CD47 is often upregulated in cancer cells, forming abundant CD47-SIRPα axes for cancer cells to escape elimination by the body. Further, CD47 upregulation is also found in patients having a fibrotic disease, atherosclerosis, or an infectious disease. See, e.g., Cui et al., *Nat. Commun.* 11, 2795 (2020); Kojima, et al., *Nature* 536, 86-90 (2016); and Tal et al., *mBio* 11 (2020). Blocking the CD47-SIRPα signaling axis promotes destruction of cancer cells and provides novel treatments for the fibrotic disease, atherosclerosis, and the infectious disease.

Nevertheless, it is a challenge to develop an effective treatment of a condition involving CD47 upregulation via inhibiting the CD47-SIRPα signaling axis as there is not clear guidance provided by publications as to how to select compounds for this treatment.

There is a need to develop an effective method of treating a condition involving CD47 upregulation.