



CARISMA to Present New Data at The Society for Immunotherapy of Cancer 35th Anniversary Annual Meeting

November 9, 2020

- Combination of CAR-M and anti-PD1 in mouse models show positive results

- New human myeloid cell engineering method may improve scaling and manufacturing

PHILADELPHIA, Nov. 9, 2020 /PRNewswire/ -- [CARISMA Therapeutics Inc.](https://www.carismatx.com), a biopharmaceutical company focused on discovering and developing innovative immunotherapies, today announced new preclinical findings accepted for presentation at The Society for Immunotherapy of Cancer (SITC) 35th Anniversary Annual Meeting (November 9 – 14, 2020). The data build on findings from CARISMA's foundational chimeric antigen receptor macrophages (CAR-M) platform that was published in [Nature Biotechnology in March 2020](https://doi.org/10.1016/j.nbt.2020.03.001).

CARISMA will be presenting new data evaluating CAR-M in fully immunocompetent animal models of solid tumors in "Chimeric antigen receptor macrophages (CAR-M) elicit a systemic anti-tumor immune response and synergize with PD1 blockade in immunocompetent mouse models of HER2+ solid tumors." These data show that CAR-M not only directly shrink tumors but reprogram the tumor microenvironment and induce long-term T cell mediated anti-tumor immunity. The new data also show that the combination of CAR-M with anti-PD1 therapy further enhanced tumor control and overall survival. These data build upon the initial findings that suggest CAR-M are a promising cell therapy approach for solid tumors and suggest that there is significant rationale for clinical exploration of CAR-M with immune checkpoint inhibition.

In "Development of an M1-polarized, non-viral chimeric antigen receptor macrophage (CAR-M) platform for cancer immunotherapy," new data based on CARISMA's delivery of CARs to macrophages shows that it does not require transduction with adenoviral vector Ad5f35. This method involves the delivery of chemically optimized mRNA through electroporation followed by induction of a durable M1 phenotype. This novel method showed efficient CAR expression, high viability, enhanced persistence compared to standard non-viral methods, potent anti-tumor function, and resistance to immunosuppressive factors. This platform has the potential to improve scaling and manufacturing and represents a significant advancement in the development of CAR-M.

"These findings are exciting because they show that there is so much more to be discovered scientifically, clinically, and from a manufacturing perspective, about the CAR-M platform," shared Michael Klichinsky, PharmD, PhD, co-inventor of the CAR-M technology and scientific co-founder and Vice President of Discovery of CARISMA Therapeutics.

The following posters will be published on the [SITC 35th Anniversary Annual Meeting portal](https://www.sitcmeeting.org) for registered attendees at 8:00 am ET on November 9, 2020, and CARISMA will be available for virtual Q&A sessions on Thursday, November 12 at 4:50-5:20 pm ET and Saturday, November 14 at 1:00-1:30 pm ET:

- Chimeric antigen receptor macrophages (CAR-M) elicit a systemic anti-tumor immune response and synergize with PD1 blockade in immunocompetent mouse models of HER2+ solid tumors
- Development of an M1-polarized, non-viral chimeric antigen receptor macrophage (CAR-M) platform for cancer immunotherapy

About CARISMA Therapeutics Inc.

CARISMA Therapeutics Inc. is a biopharmaceutical company developing a differentiated and proprietary cell therapy platform focused on engineered macrophages, cells that play a crucial role in both the innate and adaptive immune response. The first applications of the platform, developed in collaboration with the University of Pennsylvania, are autologous chimeric antigen receptor (CAR)-macrophages for the treatment of solid tumors. CARISMA Therapeutics is headquartered in Philadelphia, PA.

For more information, please visit www.carismatx.com

Media Contact:

Christina Khoury-Folkens
612-806-0757
ckhoury@w2ogroup.com

SOURCE CARISMA Therapeutics Inc.