ToolGen’s CRISPR/Cas9 gene editing platform improves T-cell anti-tumor activity in mouse model

May 22, 2018

Knockout of T-Cell suppression pathway improves T-cell anti-tumor activity in a mouse glioblastoma model

Data Presented at the American Society of Gene & Cell Therapy (ASGCT) Annual Meeting

ToolGen, Inc., a biotechnology company specializing in genome editing, today announced data demonstrating that blocking a molecular pathway that down-regulates T-cell activity with the Company’s CRISPR/Cas9 gene editing platform results in increased T-cell receptor signaling, *in vitro*, and in improved anti-tumor activity when tested against in a mouse glioblastoma tumor model. The study is being presented in a poster presentation at the American Society of Gene & Cell Therapy (ASGCT) Annual Meeting held May 16-19, 2018, in Chicago.

The poster, entitled “CRISPR/Cas9-Mediated Knockout of DGK Improves Anti-Tumor Activities of Human T Cells” (Abst. #114), examined the anti-tumor activity of T cells in a solid tumor model where immune cell function is highly limited. Diacylglycerol kinase (DGK) is an enzyme that phosphorylates diacylglycerol, an essential signal transduction molecule for T cell signaling. The DGK gene is highly expressed in tumor infiltrating lymphocytes and is responsible for inhibiting downstream signaling of T-cell receptors. Previous studies demonstrated that disruption of DGK resulted in an increase in T-cell function as well as enhanced resistance against

immunosuppressive soluble factors, such as TGF-β and prostaglandin E2. This study demonstrated that CRISPR/Cas9-mediated DGK knockout T cells improved tumor clearance in a mouse glioblastoma model.

“Therapeutic outcomes using CAR-T therapies have been disappointing when applied to solid tumors due to various immunosuppressive signals found in the tumor microenvironment,” said In-Young Jung, primary researcher at ToolGen. “High expression of DGK in tumor infiltrating lymphocytes may contribute to the immunosuppressive effect. The results from this study demonstrate that blocking DGK expression with our CRISPR/cas9 platform is a viable approach to potentially enable CAR-T-based therapies to be applied against solid tumors.”

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