GITR and Immune Function

Downstream effects of GITR activation through ligand interaction are dependent on cell type:

- On T effector cells (Teff), GITR signaling enhances survival and proliferation, thereby increasing cancer-killing activity.
- On T regulatory cells (Treg), GITR signaling blocks the suppressive abilities of Tregs, further enhancing cytotoxic T cell function.

Overall, GITR activation serves to boost the immune system’s ability to respond to threats, leading to potent anti-tumor immunity.

GITR and Cancer

In preclinical studies, activation of GITR signaling through an anti-GITR agonist enhances immunity through the activation of cytotoxic T cells and inhibition of immune-suppressive Treg activity.

Interactions with Other Pathways

By energizing the immune system, GITR signaling may synergize with other pathways to promote enhanced tumor killing activity.

About GITR

Glucocorticoid-induced TNFR-related protein (GITR) is a costimulatory activating receptor on the surface of T cells and other immune cells that functions to energize T cell responses to antigens. As a member of the tumor necrosis factor receptor (TNFR) super family of costimulatory receptors, GITR interacts with its ligand, GITRL, on neighboring immune cells.

The GITR pathway is just one of many immune pathways under investigation at Bristol-Myers Squibb. Learn more about our work in immuno-oncology by visiting:

www.immunooncology.bmsinformation.com