Editorial
Volume 10:5, 2021

Pharmaceutical Regulatory Affairs: Open Access

ISSN: 2167-7689 Open Access

STING Activation may be a New Approach to Reduce Graft-Versus-Host Disease

Harapriya Sahoo*
Department of Microbiology, Utkal University, Bhubaneswar, Odisha, India

Editorial

MUSC Hollings Cancer Center researcher identified a replacement target molecule within the fight against graft-versus-host disease (GVHD). Bone marrow transplant, a treatment surely blood cancers, is amid potentially life-threatening GVHD in nearly 50% of patients.

Recently, STING (stimulator of interferon genes) has been highly studied within the context of cancer. Data from other groups has shown that STING activation in T cells helps the immune cells fight cancer. Cancer cells are essentially a "bad" version of the body's own cells and an appropriate target for its system. In contrast, within the case of GVHD, T cells fight the body's own "good" cells - in essence, the body attacks itself. Based on the previous data, it seemed logical that prime STING activation, though good when it involves cancer, would be bad within the context of GVHD.

Researcher findings during a mouse model of GVHD confirmed this hypothesis. Within the mouse model, GVHD was induced by bone marrow transplant, which closely models the disease development in humans.

To understand how GVHD develops after bone marrow transplantation, one must consider two immune systems: the donor's and therefore the recipient's. The key immune cells are the antigen-presenting cells and therefore the T cells. The system knows what to attack supported specific "tags," called antigens, that are shown to the T cells by the specialized antigen-presenting cells. Dendritic cells are the foremost effective antigen-presenting cells, and that they play a critical role in GVHD.

Work from other research groups in cancer has demonstrated that STING signaling can regulate antigen- presenting cell function. STING is a crucial molecule during a DNA-sensing pathway that leads to the assembly of inflammatory cytokines. But it's not known how STING regulates these cells within the context of GVHD.

The researchers used the mouse models to work out whether GVHD improved or worsened when STING was 1) absent within the donor immune cells, 2) absent within the recipient immune cells and 3) overexpressed within the recipient immune cells. GVHD severity wasn't changed when STING was absent from the donor immune cells. However, GVHD was more severe and mortality rates were higher when STING was missing from the recipient immune cells.

Scientists then checked out different cell subsets to undertake and understand which cells were most impacted by the loss of STING. Surprisingly, STING expression within the recipient mouse's antigen-presenting cells (dendritic cells) reduced donor T cell expansion and migratory ability after bone marrow transplant. In other words, it made it less likely that the T cells of the recipient mouse would attack its "good" cells and cause GVHD. This finding was confirmed employing a pharmacological drug that turned on the STING molecule. Activating STING within the host before transplantation reduced GVHD severity.

The finding during a mouse model that activating STING with a pharmacological drug reduced GVHD might be clinically relevant therein it suggests the likelihood that a STING-activating drug might protect bone marrow transplant recipients from GVHD. Far more basic and clinical researches are going to be required to assess that possibility.

To understand why the research team observed what they did, they're going to still unravel the biological functions of the STING molecule. Unanswered questions include what makes STING function differently in several immune cell subsets.

How to cite this article: Harapriya Sahoo. "STING Activation may be a New Approach to Reduce Graft-Versus-Host Disease." Pharmaceut Reg Affairs 10 (2021): 250.

***Address for Correspondence:** Sahoo H, Department of Microbiology, Utkal University, Bhubaneswar, Odisha, India, E-mail: harapriyas97@gmail.com*

Received 20 May 2021; **Accepted** 25 May 2021; **Published** 30 May 2021