Editorial

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Editorial Note on Interferon and GvHD

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Editorial Note

For patients with hematological malignancies, allogeneic hematopoietic stem cell transplantation (allo-HSCT) is the most successful and curative therapy option. Graft-versus-leukemia (GvL) effects and robust immunological reconstitution are two of the life-saving advantages of all-HSCT. The majority of patients, however, suffer Graft-vs-Host Disease (GvHD), which causes organ/tissue destruction in the skin, liver, and gastrointestinal system. As a result, substantial study on GvHD has been conducted, with IFNs being identified as a therapeutic target. Despite this, the pleiotropic nature of IFNs has hampered our efforts to achieve optimal GvHD management.

The purpose of this paper is to identify the roles and mechanisms of IFNs in GvHD and to offer new treatment options for preventing GvHD while maximizing the advantages of allo-HSCT. Xenogeneic cell transplant models were used to further investigate mechanisms of primary human T cell responses in GvHD. Some researchers spoke into detail on the development of humanized mouse models and a xenogeneic transplant model system, which led to study into human T cell biology. Human T cell receptors (TCRs) have cross-reactivity with murine MHC, as well as cytokines and co-stimulatory proteins, which helps the models. They went on to discuss the significance of TCR, the capacity to detect non-self-antigens on MHC, and cellular treatment, which involves inhibiting or boosting cytokine release and co-stimulatory/inhibitory signaling pathways.

IFNs are not only vital for immune cell development and growth, but they also have a role in the GI tract. Researchers looked at the pathophysiology of GvHD, which is characterized by a cytokine network imbalance in the GI tract. In GvHD, type I and type III IFNs are critical for maintaining the intestinal epithelial barrier integrity and modulating immunological responses, according to the scientists. However, prior to allo-HSCT, the conditioning regimen destroys epithelial cells and causes the production of DAMPs and PAMPs, resulting in local inflammation, inflammatory cytokines, and APC activation. This causes the alloreactive T cells to become activated and expand, producing further tissue damage and inflammation, as well as the release of cytokines such as type II IFN and chemokine in the GI tract. The use of JAK inhibitors as a treatment for GvHD and cytokine release syndrome is a promising method. The involvement of the JAK/STAT pathway in T cell activation and expansion, APC function, and Treg expansion was discussed by the researchers. The understanding led to the notion of utilizing ruxolitinib or baricitinib to disrupt the JAK/STAT pathway in order to treat GvHD. Ruxolitinib has also been demonstrated to be effective in the treatment of GvHD patients in clinical trials. 70 Chinese patients with steroid-refractory chronic GvHD (SR-cGvHD) were treated with ruxolitinib in a retrospective analysis, and the overall response, full remission, and partial remission rates were evaluated.

Previous research suggests that recombinant human LYG1 protein (rhLYG1) can boost CD4+ T cell activation and IFN-production, limiting tumor development. Researchers investigated the role and processes of LYG1 during GvHD and speculated that it had a role in the development of GvHD. The study found that LYG1 impairment in donor T cells lowered the severity of GvHD, as predicted. In further detail, LYG1 deficiency in donor T cells suppressed CD4+ T cell activation and IFN- expression while boosting FOXP3 expression, decreasing CXCL9 and CXCL10 production as well as allogeneic CD4+ T cell infiltration into target organs. Despite the fact that LYG1 blockage only resulted in a minor increase in overall survival, our data suggest that targeting LYG1 might be a promising therapeutic strategy for reducing GvHD.

Scientists use allo-HLA across-reactivity to shed light on the possibility of off-target toxicity. It was studied if HLA-restriction, HLA background, and/or virus-specificity might predict the likelihood of allo-HLA cross-reactivity using third-party donor-derived virus-specific T cells. The results showed that HLA-B*08:01-restricted T cells exhibited the highest allo-HLA cross-reactivity independent of viral specificity, implying that choosing T cells with a specific HLA restriction and background might lower the likelihood of off-target toxicity. Overall, the paper presents an overview of current developments in IFN functions in GvHD.

This also highlighted prospective treatment options to control IFN signaling to prevent GvHD while keeping the advantages of all-HSCT, such as CD27 agonizing monoclonal antibodies, JAK inhibitors, and targeting LYG1. In order to deliver promising transplantation therapy, more study into the actual processes of IFNs in the context of transplantation is required.