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A Review on Allogeneic Bone Marrow Transplantation

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Abstract

According to clinical data, patients undergoing allogeneic hematopoietic stem cell transplantation (HSCT) are a ' the inf ammatory immune response is dysregulated in this clinical setting. Here, by utilizing a mouse model of haploidentical bone marrow transplantation (haplo-BMT), we found that uncontrolled macrophage irritation underlies the pathogenesis of the two LPS-and E.coli-prompted sepsis in bene f ciary creatures with join versus-have illness (GVHD). Macrophage-induced inf ammation was mechanistically dependent on MMP9-mediated activation of TGF-1 when neutrophil maturation was de f cient in GVHD mice following haplo-BMT. Consequently, post-haplo-BMT, adoptive transfer of mature neutrophils puri f ed from wild-type donor mice prevented infectious as well as sterile sepsis in GVHD mice. Together, our discoveries distinguish an original mature neutrophil-subordinate guideline of macrophage f ery reaction in a haplo-BMT setting and give valuable insights for creating clinical procedures for patients experiencing post-HSCT sepsis.

e of ds: Bone marrow transplantation; Neutrophil; Macrophage; Proin ammatory; Organ Dysfunction

Int f od ction

Sepsis is a life-threatening immune response to infection disorder characterized by widespread systemic in ammation and organ dysfunction. During sepsis, the primary proin ammatory cytokine-producing cells are innate immune cells, which express pattern

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and IL-12 levels in GVHD mice, which are indicators of the cytokine storm. In addition, we found that GVHD mice's serum levels of the classic immune-regulatory cytokine IL-10 were elevated indicating that these recipient animals' immune systems were not functioning properly following haplo-BMT. When compared to non-GVHD and untransplanted WT mice, the serum TGF-1 level in GVHD mice signi cantly decreased following LPS administration [8]. Septic GVHD mice had a systemic in ammatory response, as evidenced by elevated TNF- and IL-6 production in the liver, lungs, and spleens two hours a er LPS injection. When compared to macrophage-replete GVHD mice, macrophage depletion signi cantly reduced serum TNF- and IL-6 levels following LPS injection, but only slightly increased early survival. In E. coli-induced sepsis, macrophage depletion consistently increased the survival of GVHD mice a er E. coli infection and reduced the presence of proin ammatory cytokines in the sera. Because the bacterial loads in the peritoneal cavity, blood, spleen, and lung of macrophage-depleted GVHD mice were not signi cantly di erent from those of macrophage-repleted GVHD mice, there was no correlation between the improved survival rate of macrophage-depleted GVHD mice and the control of infection in primary sites of infection or bacterial propagation [Figure 1].

Res lt

It was discovered that neutrophils, a diverse population of cells, play regulatory roles in in ammatory responses. For in vivo neutrophil depletion, monoclonal antibodies against Gr-1 or Ly6G, which recognize various antigen epitopes on neutrophils, have been extensively used. Without a ecting the number of splenic macrophages, either antibody could e ectively deplete neutrophils in 24 hours in GVHD mice (Figures S2A7(v)8(er)7ese ne

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