Abstract

T cell-mediated diseases, including autoimmune disorders and graft-versus-host disease (GvHD), represent significant clinical challenges due to pathological T cell activation and proliferation. Mesenchymal stem cells (MSCs) have appeared as prospective immunomodulatory agents capable of suppressing T cell functions. This study aimed to investigate the effects and mechanisms by which MSCs modulate activated T cells. Wharton's Jelly-derived MSCs (WJ-MSCs) and amniotic fluid-derived MSCs (AF-MSCs) were co-cultured with activated murine CD4+ T cells under various stimulation conditions, including phorbol 12-myristate 13-acetate plus ionomycin (P+I), concanavalin A (ConA), and CD3/CD28-coated beads. Cytokine production (IL-2 and IFN-γ) was quantified using ELISA, and flow cytometry with eFluor 670 proliferation dye was used to assess T cell proliferation. Results showed that both MSC types significantly suppressed IL-2 and IFN-γ secretion, with AF-MSCs exhibiting a stronger inhibitory effect on T cell proliferation and cytokine production. The immunosuppressive effects could be mediated through a combination of soluble factors and direct cell-to-cell contact, potentially disrupting key intracellular signaling pathways involved in T cell activation. These findings suggest that MSCs possess strong immunomodulatory properties that could be used for treating T cell-mediated diseases. However, the limitations of the in vitro, factors distinction, and cross-species model highlight the need for further in vivo validation. Future studies should explore MSC source-specific mechanisms, differentiation between soluble factors and direct cell to cell contact, and investigate extracellular vesicle-mediated effects to enhance knowledge in this field. This research contributes valuable insights into MSC-T cell interactions and supports the development of targeted MSC-based immunotherapies.

Keywords: Mesenchymal stem cells, T cell activation, T cell mediated diseases, immunomodulation, cytokine suppression.