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MESENCHYMAL STEM CELLS: A NEW TOOL FOR IMMUNOTHERAPY?

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SUMMARY

Introduction: Stem cells are not only exploited for the restorative functions yet been utilised to support the reparative action of the host stem cells by modulating toxic inflammation. Such action of stem cells, especially mesenchymal stem cells (MSCs) creates a conducive microenvironment that enables a proper execution of the host repair system. Modulation of the immune system is crucial when the repair of the organ or system jeopardised by the inflammation, in particularly, disease that derived from unwanted immune activation such as graft-vs-host disease and autoimmunity. Thus, by using global gene expression profiling, the current study was designed to decipher the transcriptional modulation involved in suppressing T cell-mediated immune response by MSCs. **Materials and methods:** Peripheral blood mononuclear cells from the healthy donor served as a source of T cells. T cells were activated using a common stimulator, PHA and also by CD3/28 microbeads that mimic the antigen-specific stimulation. **Results:** When T cells and MSCs were cocultured at various ratio, dose-dependent inhibition of T cell proliferation was noted through cell-to-cell contact mode. The inhibition of T cells is not due to apoptosis induction, but the cells were arrested at the G0/G1 phase of cell cycle. Notably, T cells that were cultured with MSCs, shown a downregulation of IFNG, CXCL9, IL2, IL2RA and CCND3 genes while IL11, VSIG4, GJA1, TIMP3 and BBC3 genes were upregulated. **Discussion:** Based on the lymphocyte proliferation/activation, apoptosis, and cell cycle and immune response ontologies and the Ingenuity Pathway Analysis, 13 canonical pathways were identified as enriched with these dysregulated genes. The most potential pathways that been attributed to the suppression of T cells include T helper cell differentiation, cyclins and cell cycle regulation as well as gap/tight junction signalling. **Conclusion:** MSCs-mediated T cell immunosuppression is not specific where it mainly involves a pan-dysregulation of genes that participated in cell cycle and proliferation.