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Mesenchymal Stem Cells Inhibit Human Th17 Cell Differentiation and Function and Induce a T Regulatory Cell Phenotype

Soufiane Ghannam, Jérôme Pène, Gabriel Torcy-Moquet, Christian Jorgensen and Hans Yssel

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This article has a correction. Please see:

- Errata - December 01, 2013

Abstract

Mesenchymal stem cells (MSCs) exert immunomodulatory properties via the inhibition of T cell activation and proliferation. Because of the deleterious role of Th17 cells in the pathogenesis of inflammatory disease, we investigated whether proinflammatory cytokines could modify the expression of adhesion molecules on human MSCs, thereby contributing to increased Th17 cell adhesion to MSCs and, as a consequence, modulating the function of the latter cells. IFN-γ and TNF-α synergistically enhanced the expression of CD54 by MSCs, enabling the CCR6 chemokine ligand CCL20 to induce in vitro adhesion of Th17 cells to MSCs. MSCs prevented the in vitro differentiation of naive $CD4^+$ T cells into Th17 cells and inhibited the production of IL-17,

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trimethylation of the corresponding region in the RORC gene in Th17 cells. These epigenetic changes were associated with the induction of fork head box p3 and the acquisition by Th17 cells of the capacity to inhibit in vitro proliferative responses of activated $CD4^+$ T cells, which was enhanced when MSCs were preincubated with IFN-γ and TNF-α. These results showed that, under inflammatory conditions, MSCs mediate the adhesion of Th17 cells via CCR6 and exert anti-inflammatory effects through the induction of a T cell regulatory phenotype in these cells.

Footnotes

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- Abbreviations used in this paper:

ChIP
chromatin immunoprecipitation

FOXP3
fork head box p3

iNOS
inducible NO synthase

MFI
mean fluorescence intensity

MSC
mesenchymal stem cell

OSM
oncostatin M

ROR
retinoid-related orphan receptor

Treg
T regulatory.

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