









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

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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<sup>+</sup> 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-y and TNF- $\alpha$ . 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**

- S.G. was supported by grants from the Agence Nationale de la Recherche and the Société Française de Rhumatologie.
- 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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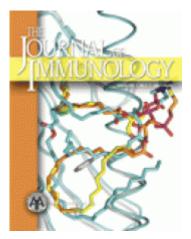
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