

The Expression of Chemokine Genes in Neutrophils Exposed to Leishmania

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Abstract: *CCL3 is a gene on chromosome 17q12 that encodes a small inducible cytokine, commonly known as macrophage inflammatory protein 1 (MIP-1) alpha, which is produced by CD8+ T cells and is a major HIV-suppressive factor. The aim of this study was to determine CCL3 gene expression in neutrophils exposed to leishmania. In this study we used Leishmania infantum parasite. Stored parasite samples were kept in nitrogen tanks. Parasites were transferred to the NNN medium. In the third and seventh days after culturing, parasite growth was examined. Expression of CCL3 gene was evaluated in neutrophils exposed to Leishmania using RTPCR. Our results showed that CCL3 gene expression non-significantly decreased in neutrophils exposed to Leishmania.*

Keywords: *CCL3, Neutrophiles, Leishmania*

1. Introduction

CCL3 is a gene on chromosome 17q12 that encodes a small inducible cytokine, commonly known as macrophage inflammatory protein 1 (MIP-1) alpha, which is produced by CD8+ T cells and is a major HIV-suppressive factor. It binds to chemokine receptors CCR1, CCR4 and CCR5. [1],[2] Neutrophil is the most abundant type of granulocytes and the most abundant (40% to 75%) type of white blood cells in most mammals. They form an essential part of the innate immune system. Its functionality varies in different animals. [3],[4] Leishmania is any of a genus (*Leishmania*) of flagellate protozoans that are parasitic in the tissues of vertebrates; an organism resembling the leishmanias that is included in the family (Trypanosomatidae) to which they belong[5],[6] The aim of this study was to determine CCL3 gene expression in neutrophils exposed to leishmania.

2. Material And Methods

In this study we used *Leishmania infantum* parasite. Stored parasite samples were kept in nitrogen tanks. Parasites were transferred to the NNN medium. In the third and seventh days after culturing, parasite growth was examined. Expression of CCL3 gene was evaluated in neutrophils exposed to *Leishmania* using RTPCR.

3. Results

Our results showed that CCL3 gene expression non-significantly decreased in neutrophils exposed to *Leishmania*.

4. Discussion

Studies show high prevalence and spread of leishmaniasis in many regions.[7]-[9] The Leishmaniasis serious clinical complications include skin lesions which impose considerable cost burden for its treatment.[10],[11] So, any study regarding to finding the mechanisms involved in Leishmania function specially on immune system is important. On the other hand, C-C motif ligand 3 (CCL3) chemokine plays a crucial role in the inflammation process, cell migration and chemoattraction of monocytes/macrophages, neutrophils and mast cells. CCL3 is overexpressed by malignant cells in B-cell disorders, including chronic lymphocytic leukemia and multiple myeloma [12]; however, in our study we did not find CCL3 gene expression alteration in neutrophils exposed to Leishmania in culture medium.

5. Conclusion

Our results showed that leishmania parasite has not stimulated the CCL3 gene expression in neutrophils exposed to Leishmania.

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