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TLR signals modify the expression of scavenger receptors

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Introduction

Toll like receptors (TLRs) and scavenger receptors are expressed on the surface of monocytes in order to participate in relevant innate immune functions. It has been established that TLRs can discriminate molecular structures normally absent in the healthy host, whereas CD36 is a class B scavenger receptor that can coordinate responses to TLR2:TLR6 agonists. Although recent studies have focused on CD36-TLR cooperation in mediating phagocytosis, it is not know whether TLRs can regulate the expression of scavenger receptors.

Aim

To understand how TLR signals can interact with scavenger receptors, we analyzed the expression of CD36 on monocytes after the stimulation with TLR4 (LPS) and TLR2 (Pam3CSK4 and FSL1) ligands.

Methods

Human peripheral blood mononuclear cells (PBMCs) from healthy donors were incubated with Pam3CSK4, (TLR2:TLR1 ligand) FSL1 (TLR2:TLR6 ligand) and ultrapure LPS (TLR4 ligand). After 24, 48 and 72 hours of culture, CD36 expression was analyzed by flow cytometry and cytokine production in the supernatants was determined by ELISA.

Results

After culturing with each TLR ligand, CD36 expression was downregulated on CD14+ monocytes, being LPS the ligand with the strongest effect. The kinetics analysis revealed that 48h was the peak of downregulation, with the recovery of CD36 expression at 72h in Pam3CSK4 and FSL1 cultures. Since the binding of TLR ligands induced cytokine production and secretion, we analyzed whether these cytokines were responsible for the observed downregulation. TNFalpha and IFNgamma (at lesser extent) but not IL-10 were able to downregulate CD36 expression. Blocking specifically the TNF effect after TLR binding with neutralizing antibodies, CD36 downregulation was partially abrogated. While after TLR2 or TLR4 binding, CD36 was downregulated by internalization, other scavenger receptors or molecules involved in the TLR ligand recognition were not modulated.

Conclusion

Since CD36 is required for TLR responses, the TLR-induced downregulation of CD36 could operate as a regulator of the innate immune response through TLRs.

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