عنوان مقاله:

Assessment of CD44, SYNDECAN and Integrin Gene Expression in Mesenchymal Stem Cells (MSCs) After Priming by Poly (I:C)) as a Synthetic Analog of Double-Stranded RNA (dsRNA) in Order to Effective Type 2 Interaction

محل انتشار:

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خلاصه مقاله:

Background and Aim: The use of MSCs for cell therapy relies on thecapacity of these cells to recruit, homing and engraft into the target tissue.Homing of MSCs can occur but does so with only poor efficiency. Thus,the efficiency of MSCs transplantation is limited by lower homing ofMSCs. MSCs possess immunoregulatory properties due to the expressionof the key components of innate immunity such as TLRs. The aim of thepresent study was to evaluate the role of TLR3-primed MSCs on mRNAgene expressions of several CAMs that involved in stem cell homing.Methods: At first, to quantify the impact of the TLR3 agonist poly (I:C) onthe mRNA expression levels of TLR3 in hMSCs, we performed RT-PCRand real-time RT-PCR assays. RT-PCR analysis revealed that 4 h exposureto poly (I:C) elevated TLR3 mRNA expression in hMSCs in a concentrationand time-dependent manner. Real-time RT-PCR showed that TLR3 mRNAlevels reached the highest amount in MSCs exposed to 5 µg/mL poly (I:C)for 4 h during different exposure times. Also, the incubation with 5 µg/mL poly (I:C) for 4 hours preferably elevated proinflammatory cytokinesmRNA levels.Results: Here, we show that exposure to the TLR3 agonist poly ((I:C))increased the mRNA expression levels of TLR3 and cell adhesionmolecules such as CD44, Syndecan and integrin isoforms such as α1β1,α2β1, α3β1, α5β1 and αvβ5. Poly(I:C) exposure elevated intracellularsignaling pathway that associated with TLR3 signaling including TRIF,TRAF-3, NF-kB and IκB and decreases proinflammatory cytokines such asIL-6. On the other hand, TLR3 agonist prompted an expression of integrinisoforms and integrin-mediated adhesion molecules that involved incell-cell interaction and homing. In addition, we observed that other celladhesion molecules such as CD44 and Syndecan were significantly upregulatedin response to TLR3 priming of BM-MSCsConclusion: TLR3 priming may enhance MSCs to the expression ofseveral cell adhesion molecules that involved in MSCs recruitment,mobilization, homing and retention. Our findings not only clarify thenovel signaling cascade from TLR3-priming to immunoproperty processbut also implicate potential targets for genetic and pharmaceuticalmanipulation in MSCs-based therapy for increasing efficiency ofrecruitment and homing of MSCs for future clinical applications.

کلمات کلیدی:
TLR3; Innate immunity; MSCs; Homing