The contributions of previous research on the benefits and future treatments of magnesium as an implant material

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Introduction:

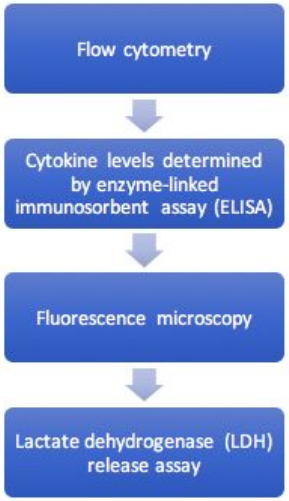
William Hunter proposed that damaged cartilage cannot be reconstituted. There is a more extensive availability of mesenchymal stem cells (MSC's) highlights the attractiveness of their use in cartilage regeneration. After investigating the effects of magnesium on the nuclear translocation of nuclear factor-κB (NF-κB) induced by LPS and IFN-γ in RAW 264.7 (RAW) cells to validate its anti-inflammatory mechanism as well as the investigation of the chondrogenic differentiation of human bone marrow MSCs (hBMSCs) co-cultured with activated macrophage cell-conditioned medium and the potential effects of magnesium addition in the process, the following conclusion can be drawn:

The use of Magnesium showed evidence of enhancing the chondrogenic differentiation of mesenchymal stem cells by inhibiting activated macrophage-induced inflammation.

Purpose:

To examine the potential effects of magnesium on the phenotypic changes in macrophages and their release of inflammatory cytokines with or without lipopolysaccharide (LPS) and interferon-γ (IFN-γ) activation.

Methods:



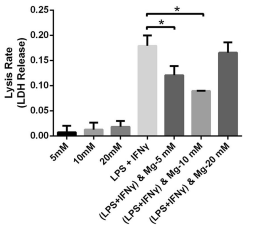


Figure 1. Lysis rates of RAW cells treated with magnesium in the absence or presence of LPS and IFN-γ for 3 days were measured by LDH release assay. (**p < 0.01; ***p < 0.001). (*Hu et al. Scientific Reportsvolume 8, Article number: 3406 (2018)*)

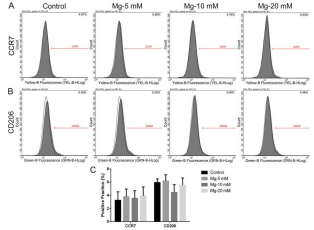


Figure 2. The effects of magnesium on the phenotype switch of non-activated macrophages. (A and B) Representative histograms of flow cytometric results: percentages of CCR7- or CD206-positive cells, representing M1 or M2 macrophages, respectively. The isotype controls are shown as none-filled histograms. (C) Statistical results for CCR7- or CD206-positive macrophages from three repeated experiments. (*Hu et al. Scientific Reportsvolume 8, Article number: 3406 (2018)*)

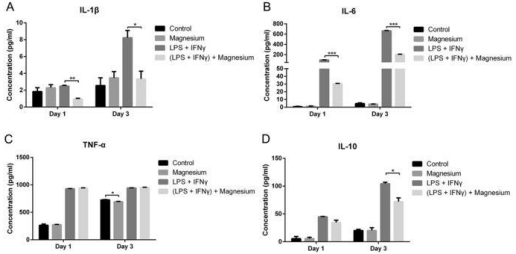


Figure 3. ELISA results for inflammatory cytokine production by RAW cells at days 1 and 3: (A) IL-1β; (B) IL-6; (C) TNF-α; (D) IL-10. (*p < 0.05; **p < 0.01; ***p < 0.001).(*Hu et al. Scientific Reportsvolume 8, Article number: 3406 (2018)*)

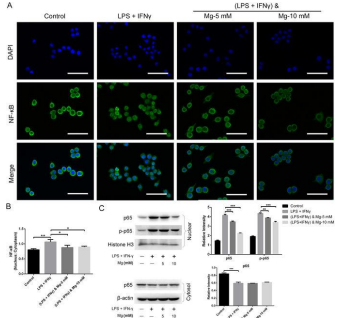


Figure 4. Effects of magnesium on nuclear NF-κB translocation in RAW cells activated by LPS and IFN-γ. (A) Nuclear translocation of NF-κB was observed by fluorescence microscopy. Scale bar = 50 μm. (B) The nuclear:cytoplasmic ratio of p65 was measured as a relatively quantitative evaluation of NF-κB nuclear translocation. (C) Immunoblotting for NF-κB p65 and phosphorylated NF-κB p65 (p-p65) was performed using the cytoplasmic and nuclear fractions. The bar chart shows the quantitative evaluation of protein bands by densitometry and the levels are presented as the mean ± SD. (*p < 0.05; **p < 0.01). (*Hu et al. Scientific Reportsvolume 8, Article number: 3406 (2018)*)

Results:

Results elucidate a novel immunoregulatory pathway for magnesium in chondrogenesis and provide foundational data for the application of magnesium-containing implants in cartilage regeneration to achieve better clinical outcomes.

Discussion:

The use of magnesium as an implant material represents a promising approach to cartilage regeneration due to its mechanical properties as well as its biodegradability. The present study investigated the effects of magnesium on macrophage polarization and the release of inflammatory cytokines from macrophages, with or without LPS and IFN-γ activation, and further elucidated whether these effects impact macrophage-induced cartilage regeneration.
After stimulation with LPS and IFN-γ, IL-1β, IL-6 and IL-10 levels were notably increased and then inhibited by the addition of magnesium.
Results show that magnesium inhibition exerted effects on activated macrophages induced by LPS and IFN-γ and the secretion of inflammatory cytokines by these macrophages, potentially contributing to the anti-inflammatory properties of magnesium. Furthermore, magnesium enhanced the chondrogenic differentiation of MSCs by inhibiting the adverse effects of activated macrophage-induced inflammation.

Future Studies:

Compare the findings that contradict when magnesium is beneficial for inflammatory response versus when it becomes dangerous for human health. Re-test the findings to validate whether magnesium is beneficial at those times from previous studies to current studies.

References:

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