

Platelet-rich plasma: a promising dose-dependent treatment for osteoarthritis pain

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Introduction

- Osteoarthritis (OA) is a highly prevalent joint disease associated with age and activity
- Platelet-rich plasma (PRP) is a therapy that utilizes concentrated blood samples to promote healing
- Dosage-dependent improvements in pain assessments following PRP treatment
- Inhibition of key inflammatory pathways such as NF- κ B and modulation of inflammatory mediators IL-1 β and TNF- α
- Disagreements persist regarding the effects on metalloproteinases (MMPs), tissue inhibitors (TIMPs), and related enzymes, which play a crucial role in joint degradation

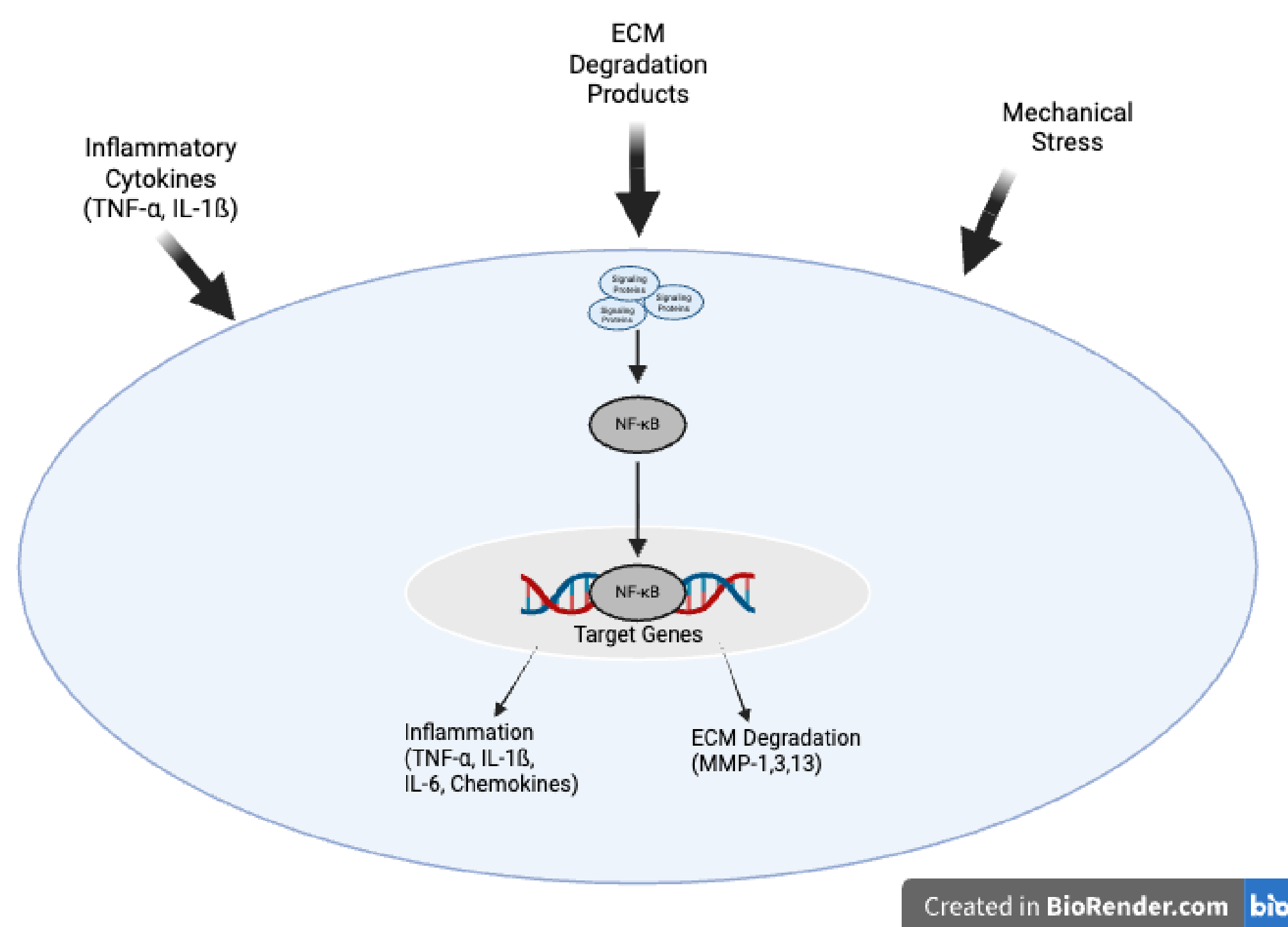


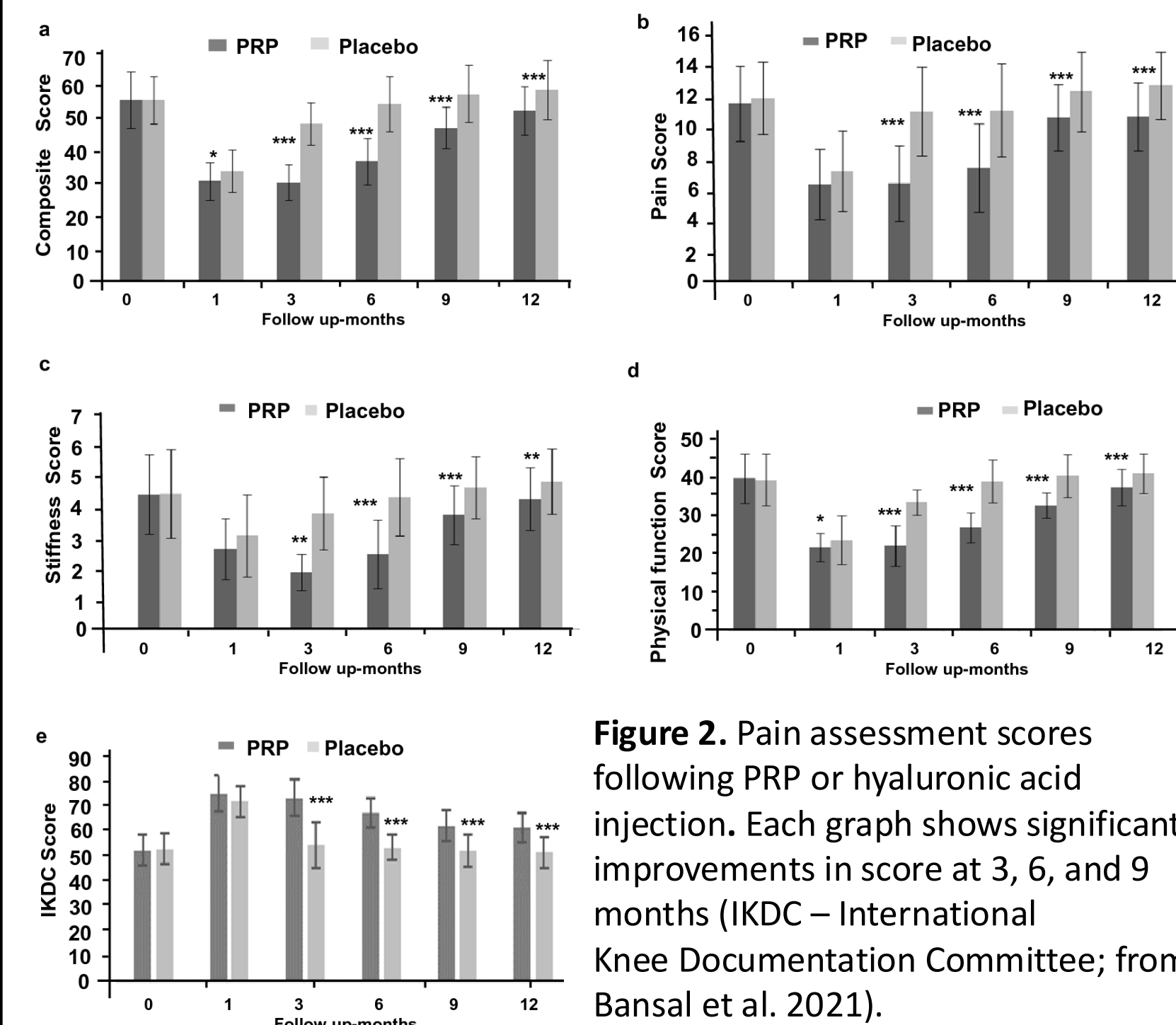
Figure 1. Adapted from Marcu et al. 2020 illustrating the NF- κ B pathway and its activation mechanism via mechanical stress and inflammatory cytokines (IL-1 β and TNF- α).

Methods

A literature review was conducted using primary sources to evaluate evidence regarding the dosage considerations, potential mechanisms of symptom management, and clinical efficacy of PRP injections.

Dosage Dependency

- Several studies, including a meta-analysis, have highlighted the importance of higher platelet dosages, with improvements observed in both pain and functionality assessments
- Earlier studies reporting minimal improvement in pain scores have been limited in the platelet dosages used



Mechanisms of Action

- Diminishes IL-1 β induced inhibition of type II collagen and aggrecan expression
- Decreases activation of NF- κ B pathway induced by IL-1 β and phenotypic changes within synovial macrophages
- One study reported decreases in TNF- α and IL-1 β accompanied by improvements in pain and functional outcomes
- Multiple studies have reported reductions in MMPs; however, discrepancies exist regarding the effects of PRP on specific MMPs and their tissue inhibitors (TIMPs).

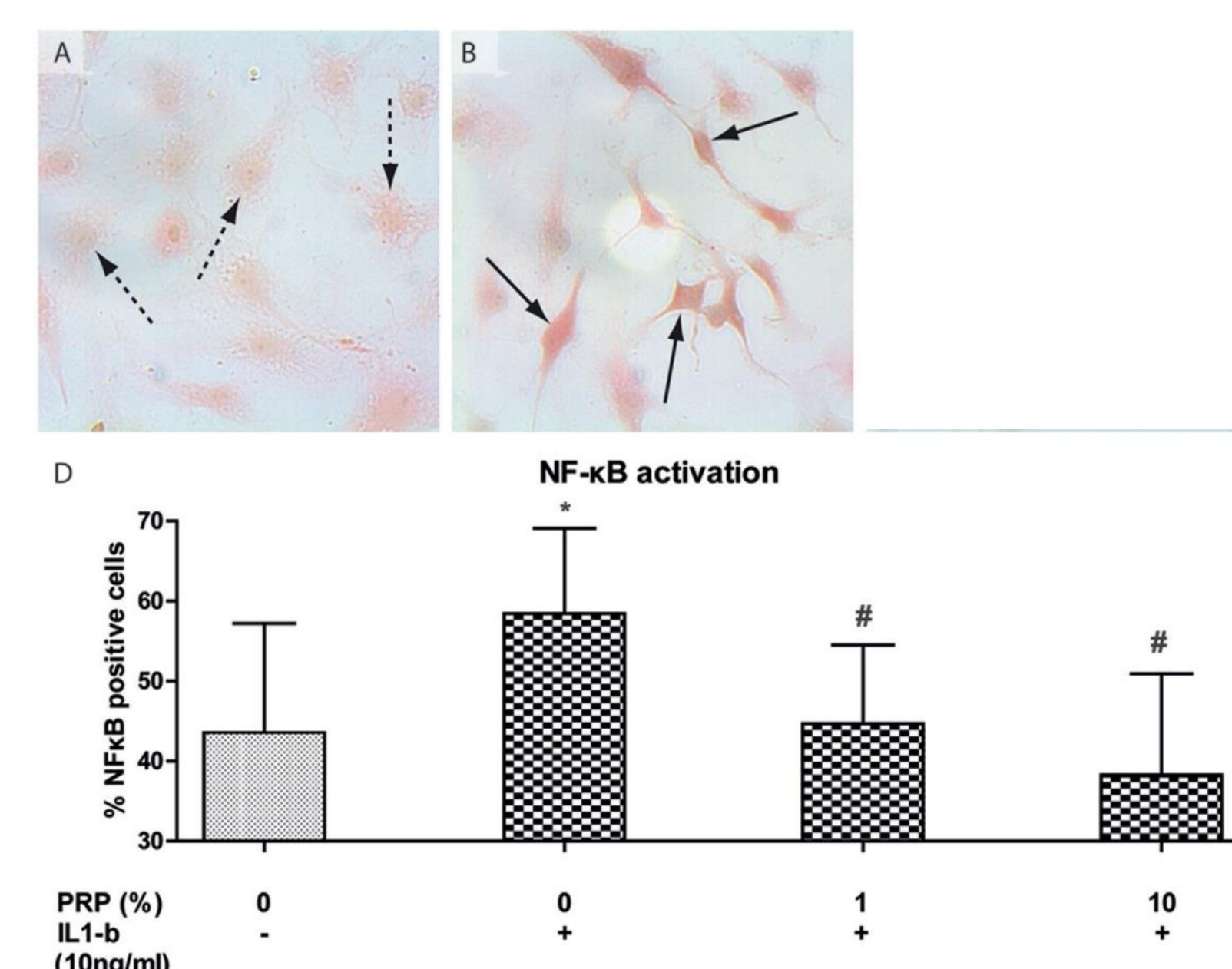


Figure 4. The effect of PRP treatment on IL-1 β induced NF- κ B pathway activation. Panel A and B shows IL-1 β treated chondrocytes before and after 10% PRP treatment (van Buul et al. 2011).

Future Directions

- Future studies should monitor inhibition patterns within the NF- κ B pathway and phenotypic changes within human synovium and cartilage
- More research is needed to determine the effect of PRP on matrix-degrading enzymes such as MMPs and TIMPs
- Build upon findings that suggest PRP promotes the formation of proteoglycan and type II collagen

Discussion

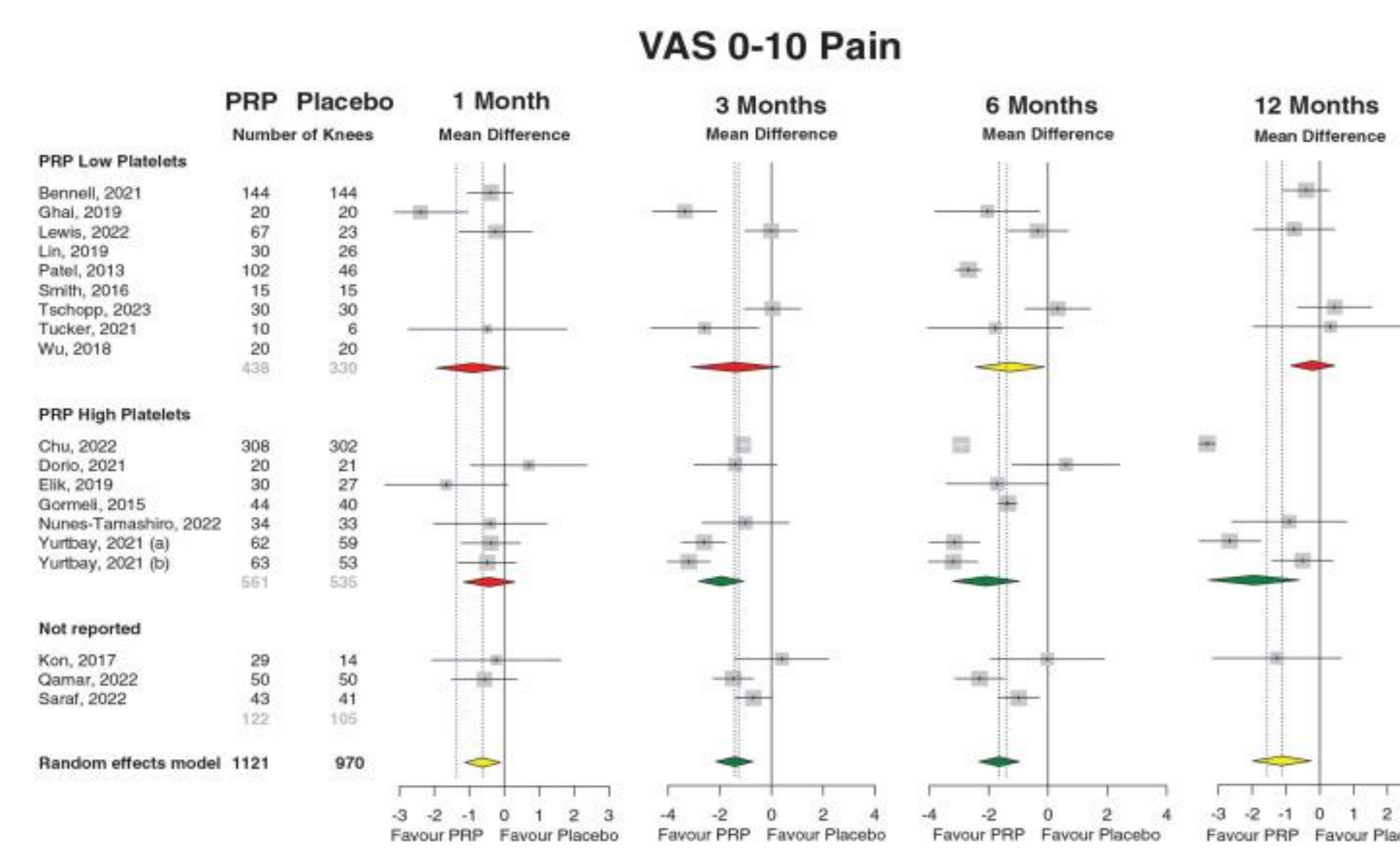


Figure 3a. Results from a meta-analysis comparing the effects of PRP on VAS assessments. The studies demonstrate a dose-dependent improvement as no meaningful improvements were seen in low-platelet PRP (VAS – Visual Analog Scale; from Bensa et al. 2017).

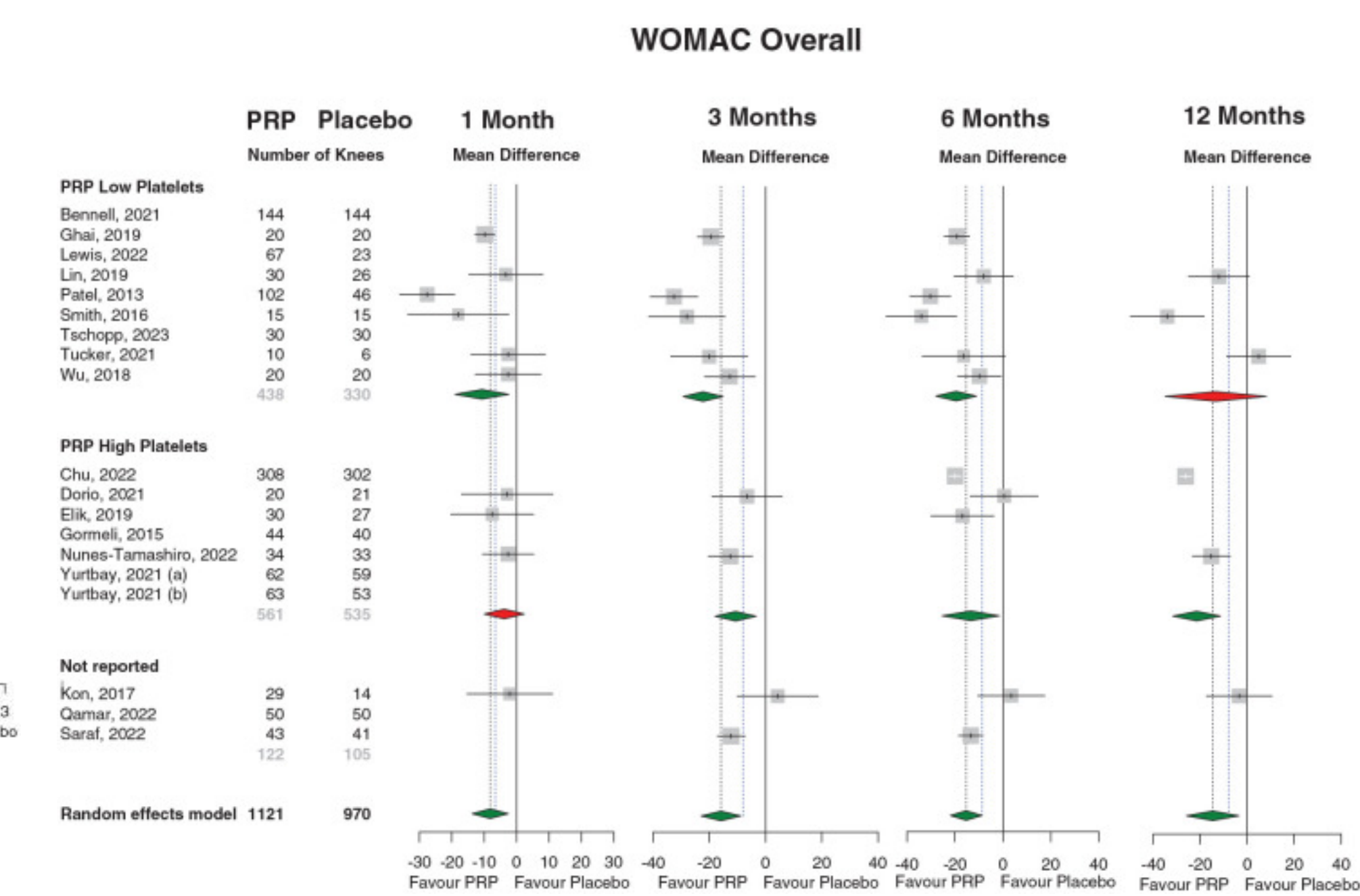


Figure 3b. Results from a meta-analysis comparing the effects of PRP on WOMAC scores. Significant improvements were seen in both low- and high-platelet PRP dosage studies (WOMAC – Western Ontario and McMaster Universities Osteoarthritis Index; from Bensa et al. 2017).

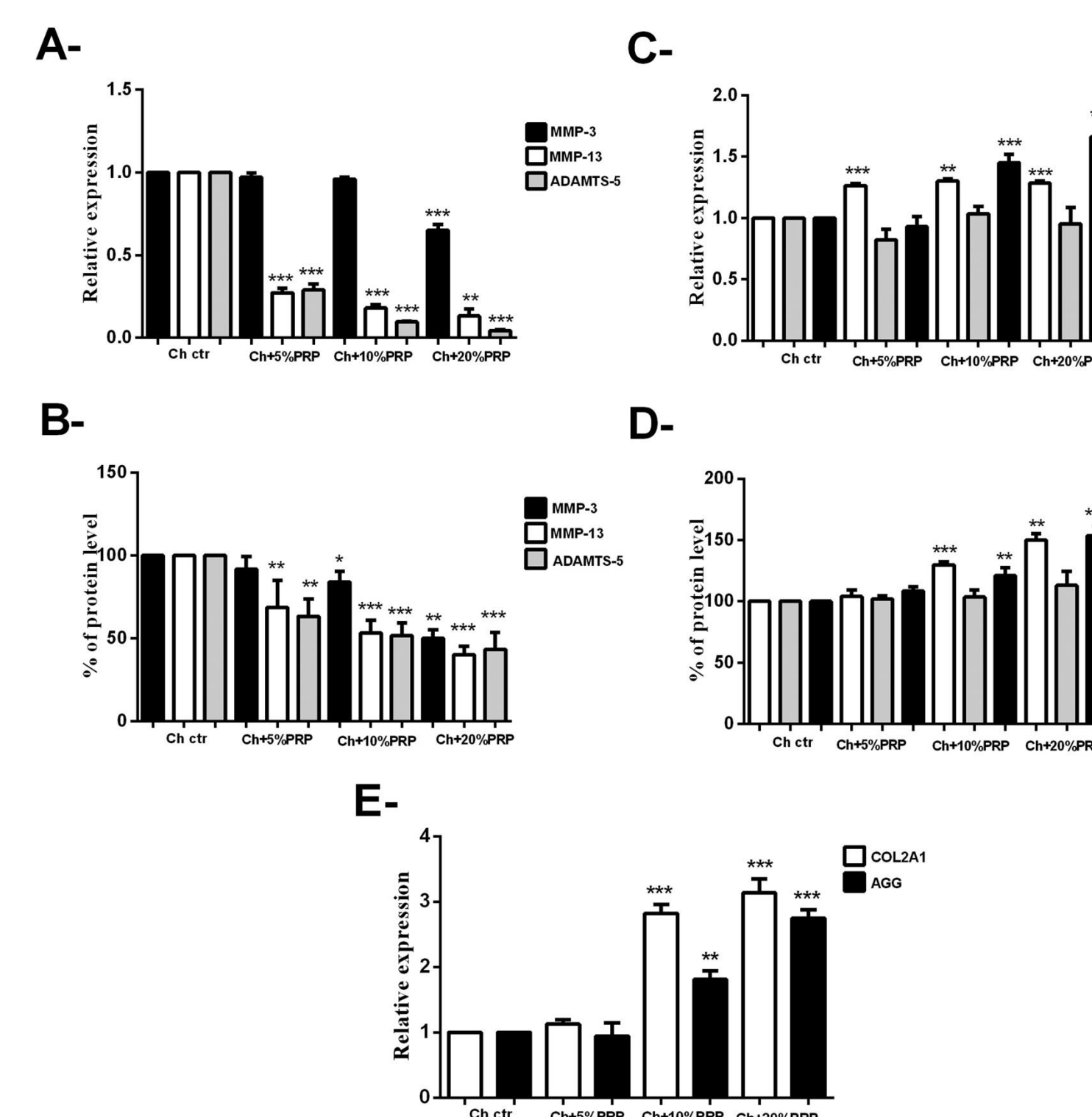


Figure 5. Relative expression and overall protein level of MMPs and their inhibitors following PRP treatment. Significant decreases found in MMPs along with increases in aggrecan and type II collagen levels (Moussa et al. 2025).

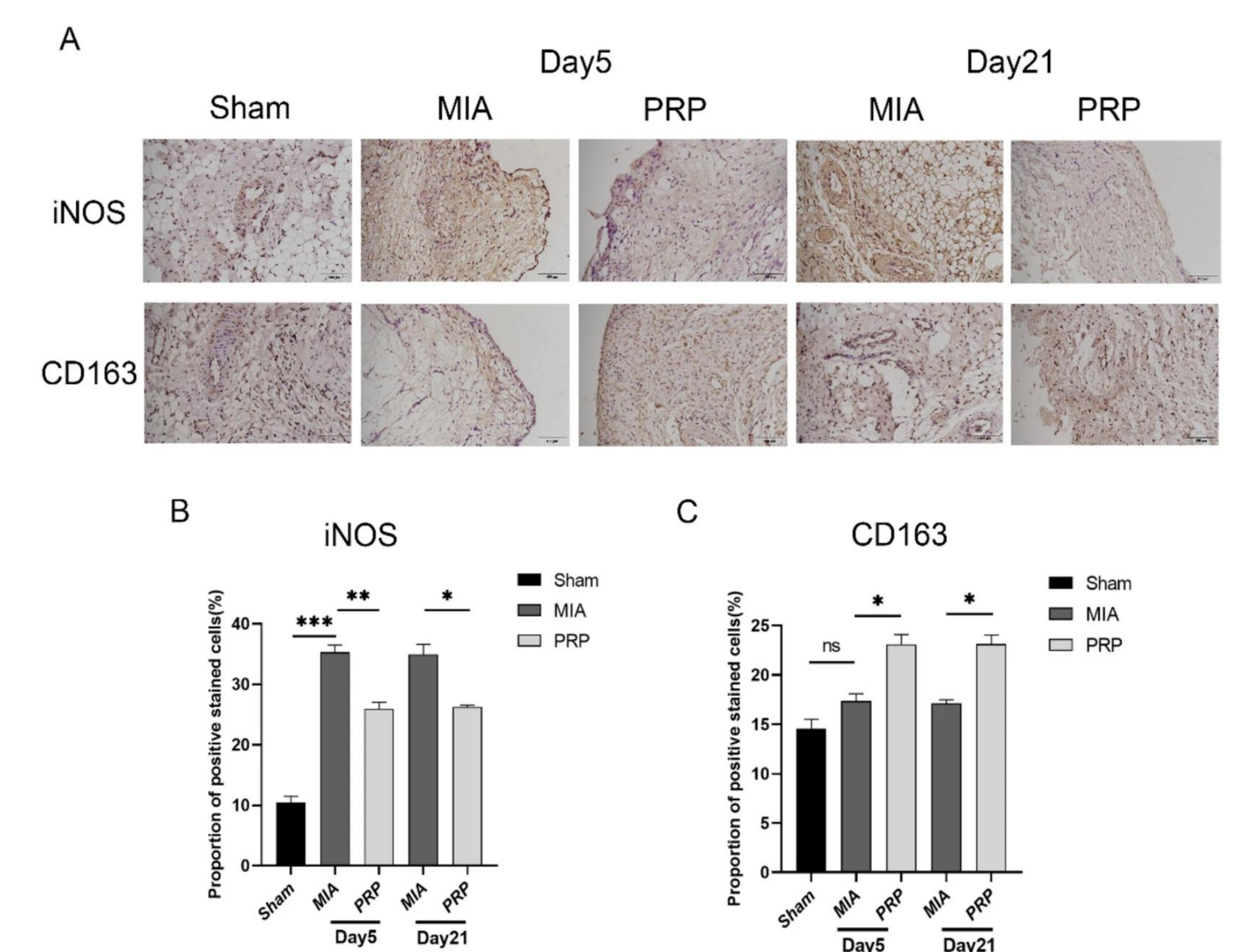


Figure 6. Comparison of iNOS and CD163 (M1 and M2 macrophage markers, respectively) immunohistochemical expression in rats. PRP treatment significantly decreased the number of iNOS-positive cells while increasing the number of CD163-positive cells (Xu et al. 2025).

References

