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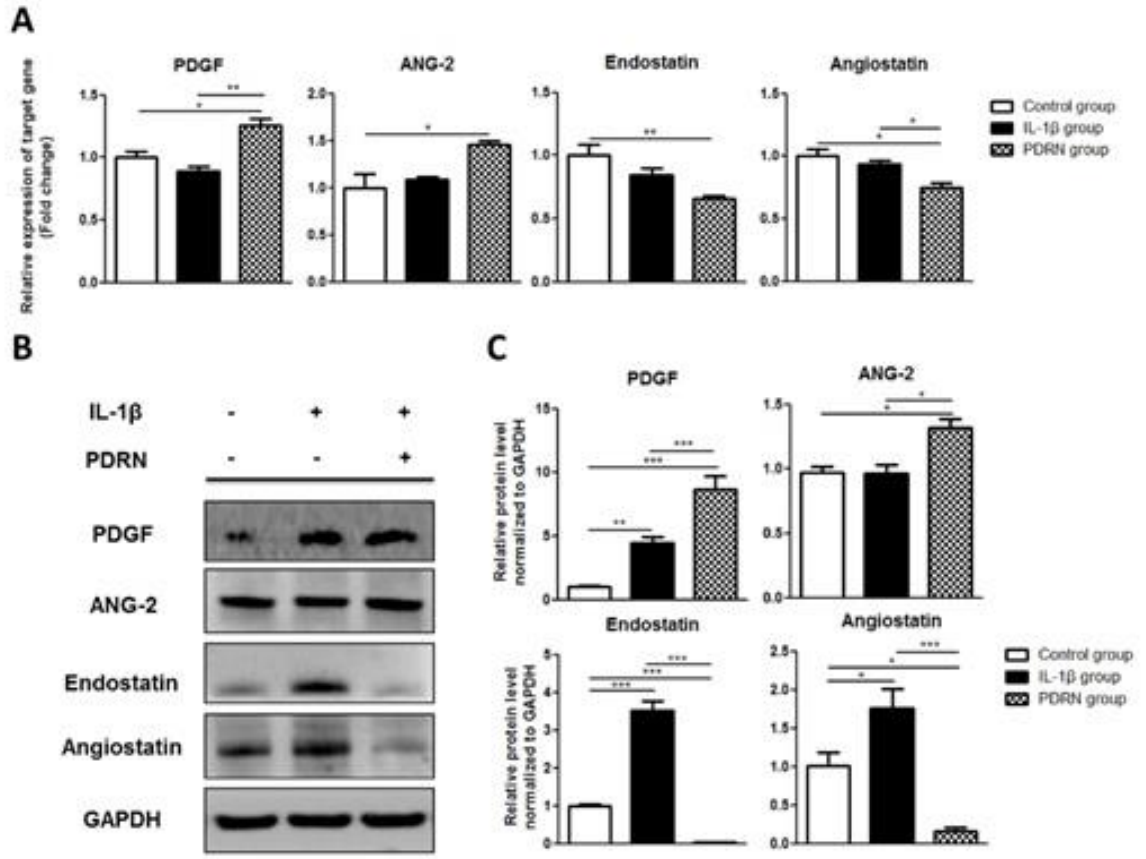
Angiogenesis protein expression in polydeoxyribonucleotide (PDRN) treated osteoarthritis cell model

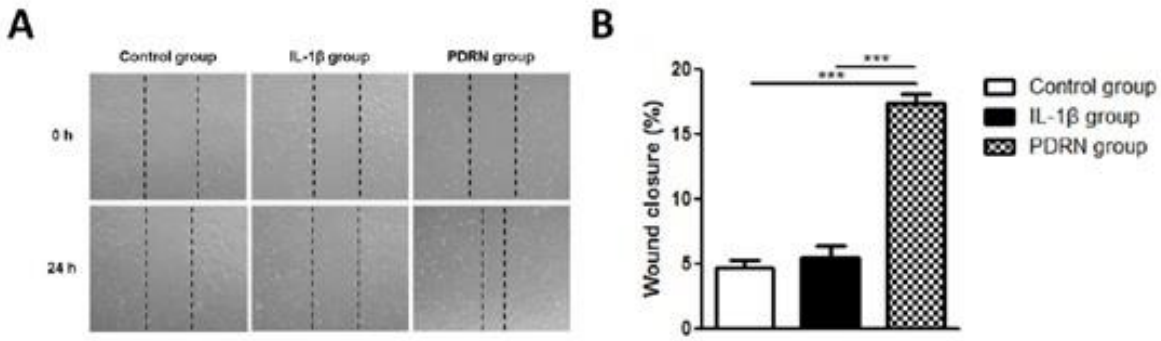
Yoon Kim[2*], Ahreum Baek[1], Sang Hoon Shin[1], Sung-Rae Cho[1†], Sang Chul Lee[1†]

Yonsei University College of Medicine, Department of Rehabilitation Medicine and Research Institute[1], Samsung Medical Center, Department of Physical and Rehabilitation Medicine[2]

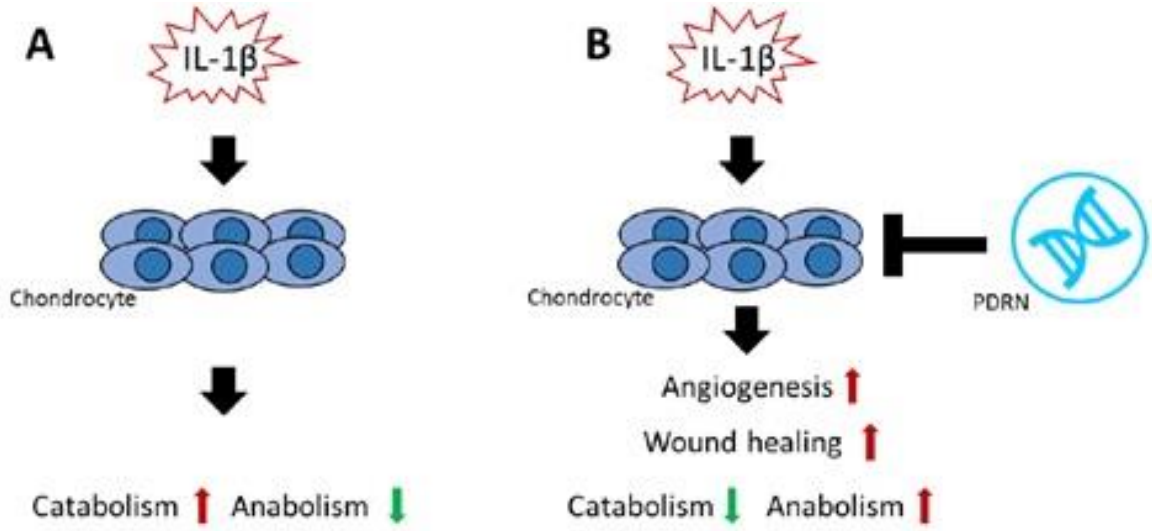
The purpose of this study was to investigate the effect of polydeoxynucleotide (PDRN) on factors associated with angiogenesis after administration of PDRN in osteoarthritis (OA) cell model. Interleukin (IL)-1β or phosphate buffered saline (PBS) was used to treat human chondrocytic cell line in hypoxic condition for 24 h (IL-1β group or control group). PDRN was then used to treat IL-1β group cells for 24 h (PDRN group). Angiopoietin-2 (ANG-2), platelet-derived growth factor (PDGF) related to pro-angiogenesis and angiostatin and endostatin related to anti-angiogenesis were chosen by Label-based Human Antibody Array 1000 for further validation studies. Quantitative real-time reverse transcription polymerase chain reaction and western blot analysis validated that levels of PDGF and ANG-2 were significantly increased in the PDRN group compared to those in the control group or the IL-1β group. However, levels of endostatin and angiostatin were significantly decreased in the PDRN group compared to those in the control group or the IL-1β group. In in vitro scratch assay, wound closure was significantly increased in the PDRN group compared to that in the control group or the IL-1β group. Moreover, PDRN decreased expression of MMP13 (a catabolic factor for OA) but increased expression of aggrecan (an anabolic factor for OA). These data suggest that PDRN may promote angiogenesis and wound healing via down-regulation of catabolism and up-regulation of anabolism in OA cell model.



Effects of PDRN on mRNA and protein levels of angiogenesis.



Effects of PDRN on cell migration. (A) Representative data of wound healing experiment. The beginning of the experiment is before treatment with PDRN and indicated as 0 h. After treatment with PDRN for 24 h is indicated as 24 h. (B) The area of the wound closure was quantified, and the ratio of wound closure was expressed as a percentage of recovered wound compared to the area at 0 h of each groups. All results are expressed as mean ± SEM. *p < 0.05; ***p < 0.001.



Effects of PDRN on in vitro OA model. (A) IL-1β induces the pathogenesis of OA in chondrocytes through up-regulation of catabolism and down-regulation of anabolism. (B) PDRN inhibit the pathogenesis of OA via up-regulation of angiogenesis and wound healing.