beneficial in the treatment of degenerative musculoskeletal problems. The purpose of this study is to evaluate PRP treatment efficacy on degenerated cartilage.

**Objectives:** In this study, we aimed to determine the efficacy of platelet-rich plasma (PRP) on mechanically damaged chondrocyte cells by using different dose, different duration of exposure and different methods of activation of platelet.

**Methods:** Human source chondrocytes (CHON-011 ATTC CRL-2846) were used in the study. Chondrocyte cells were produced in appropriate medium and an experimental cartilage model was created. The platelet-rich plasma was produced from platelets obtained by apheresis in the laboratory, from blood of volunteer. The platelet-rich plasma was adjusted at five different doses as 4.8x10^5, 2.4x10^6, 1.2x10^6, 6x10^5, 3x10^5. The first group of platelet rich plasma was left intact, the second group was detonated within seven minutes by applying ultrasound waves in water, the third group was activated with calcium chloride and the fourth group was determined as control group. Using a ten microliter pipette tip, a linear damage to theopposite side was created at the widest part of the well. Cell migration was monitored at 0-4,8-24 and 48 hours at x10 magnification by in vitro microscopy and wound healing was evaluated by photographing. Migration intervals were determined quantitatively using the program name Image J.

**Results:** When the rates of recovery were compared to the groups, no significant improvement was observed in the intact and detonated platelet groups at 4-8 and 24 hours compared to the control group. In the third group which was activated with calcium, no significant improvement was observed in all doses at 4 and 8 hours compared to the control group. However, at the 48th hours there was a significant improvement in the doses of 1.2x10^6, 2.4x10^6 and 4.8x10^6 compared to the control group (p < 0.0001). There were significant differences in intact and detonated platelets at 3x10^5 and 6x10^5 doses at 48th hours compared to control group (p < 0.0001). Significant improvement was observed in all groups at levels of 1.2x10^5 and a bove (p < 0.0001). When evaluated in terms of activation, there was a significant improvement in the exploded and intact groups at the 48th hour, compared to the calcium-activated group at doses of 3x10^5 and 6x10^5 (p < 0.01).

**Conclusion:** Cartilage damage is the main pathology in the pathogenesis of osteoarthritis. All doses of PRP used in the study contributed to improvement. Meanwhile, the most critical parameter for platelet migration was timing and significant improvement was started after 48 hours.
glutathione peroxidase activity (GPx), and inflammatory progenitor ICE/caspase-1 (ELISA) were determined.

**Results:** SAC increased OAC’s proliferation rate and adhesion profile at relatively low concentrations (1, 10 and 100 nM), but inhibited at higher concentrations (1–100 μM). SAC (1 nM–10 μM) inhibited ROS, LPO and 3-NIT, but not HNE- and AGE-modified proteins levels. SAC increased GPx but drastically down regulated ICE/caspase-1, indicating a potential redox regulating and anti-inflammatory effect.

**Conclusion:** Results suggest that SAC has favourable effects on OA chondrocytes through protecting proliferation capacity and ameliorating redox-mediated inflammatory pathways. Further studies are needed to investigate its therapeutic potential in patients with OA.

**REFERENCES**


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**THE ROLE OF CD70 IN THE PATHOGENESIS OF RHEUMATOID ARTHRITIS**

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**Background:** Rheumatoid arthritis (RA) is characterized by inflammation and cellular proliferation in the synovium. Activated lymphocytes and proinflammatory molecules are important in the pathogenesis of RA.

CD70 belongs to the tumor necrosis factor (TNF) ligand superfamily and is typically present on activated B and T lymphocytes, natural killer cells and mature dendritic cells.

CD70 expressing CD4+ T cells are enriched in the peripheral blood and synovial fluid of patients with RA and promote autoimmunity via co-stimulatory CD70-CD27 interaction.

**Objective:** In this study, we examined the presence of CD70 on the surface of fibroblast-like synoviocyte (FLS) of patients with RA (RA-FLS) and investigate the role of CD70 in the pathogenesis of RA associated with HIF-2α.

**Methods:** RA FLS were obtained from 7 patients with RA who were undergone operation like total knee replacement or synovectomy. All patients were fulfilled the 2010 ACR-EULAR classification criteria for RA.

**Results:** CD70 and HIF-2α mRNA in the RA-FLS were elevated after treatment with IL-17 and TNF-α (Figure 1, 2).

**Conclusion:** We identified the expression of CD70 on the surface of RA-FLS. And in inflammatory conditions like stimulation with IL-17 and TNF-α, both CD70 and HIF-2α mRNA were increased. The level of CD70 on the surface of RA-FLS also elevated by treatment with IL-17 and TNF-α.

**Conclusion:** We identified the expression of CD70 on the surface of RA-FLS. And in inflammatory conditions like stimulation with IL-17 and TNF-α, both CD70 and HIF-2α mRNA were increased. The level of CD70 on the surface of RA-FLS also elevated by treatment with IL-17 and TNF-α.

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