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Study of P53 in peripheral blood and synovial mononuclear cells of rheumatoid arthritis and osteoarthritis patients and its relation to the degree of disease activity

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Over expression of P53 has been described in many inflammatory conditions including rheumatoid arthritis (RA) and osteoarthritis (OA) as a protective mechanism to induce apoptosis of synovial cells. Lack of P53 function through mutation in human synoviocytes increases the development of normal synovial fibroblasts into transformed aggressive synovial fibroblasts. P53 levels were determined in supernatant of cultured mononuclear cells (MCs) isolated from peripheral blood (PBMCs) of patients with RA (n = 10) and OA (n = 10) as well as 10 normal healthy controls (C). P53 levels were also determined in supernatants of MCs isolated from synovial fluid (SFMCs) of RA and OA patients. Results of this work revealed that P53 level was significantly higher in PBMCs supernatant of RA group than those of both (C) and (OA) groups (P = 0.022). P53 level was non-significantly higher in SFMCs supernatant of RA than OA group. Significantly higher levels of P53 was detected in SFMCs culture supernatant than that of PBMCs within each RA (P = 0.003) and OA (P = 0.001) group. Results also showed a significantly positive correlation between P53 levels (in both PBMCs and SFMCs) and the disease activity score (DAS) in RA group (P = 0.01, P = 0.02 respectively) while insignificantly positive correlations between P53 level (in both PBMCs and SFMCs) and radiological grading of OA group were obtained. These results indicate that mutations and consequent dysfunction of P53 gene may result in chronic inflammation and hyperplasia in RA patients. In conclusion, gene therapy targeting P53-dependent pathway could be a promising therapy for RA and OA diseases.