# INTERLEUKIN-7 AFFECTS THE T CELL-DEPENDENT OSTEOCLAST FORMATION IN AN *IN VITRO* MODEL DERIVED FROM PSORIATIC ARTHRITIS PATIENTS

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Psoriatic arthritis (PsA) is an inflammatory joint disease. A notable propensity for aggressive bone erosions in PsA is well recognized and is manifest radiographically as dramatic jointspace loss, large eccentric bone lesions, pencil-in-cup erosions, and acrolysis (extensive resorption of the distal phalanges). In PsA, periarticular bone mineralization is maintained and there is often concomitant new bone formation in the form of periostitis and frank ankylosis. The presence of marked bone resorption coupled with adjacent new bone formation (often in the same digit) suggests a disordered pattern of bone remodeling in the psoriatic joint. Using an *in vitro* osteoclastogenesis model consisting of unstimulated peripheral blood mononuclear cells (PBMC) from PsA patients, we show, for the first time, that osteoclasts (OCs) develop spontaneously in a T cell-dependent way. Differently, in T cell-depleted PBMC cultures, the addition of M-CSF and RANKL is necessary to OC formation. Next we demonstrate the overproduction of RANKL and TNF$\alpha$, at both mRNA and protein levels, by freshly isolated T cells from peripheral blood of PsA patients. Moreover, knowing that IL-7 induces bone loss *in vivo* by induction of RANKL and TNF$\alpha$ from T cells, we show that in our system anti-IL-7 antibody inhibited osteoclastogenesis in a dose dependent manner. We also demonstrated that freshly isolated B cells from PBMCs of PsA patients were the source of IL-7 in our model. B cells in fact overexpressed IL-7 at mRNA and protein levels, and this production was up-regulated by IL-6. The potential involvement of IL-7 in the pathophysiology of PsA is supported by the *in vivo* finding of higher IL-7 levels in the sera of PsA patients than in healthy subjects. In conclusion, our findings indicate that IL-7 have a key role in the spontaneous osteoclastogenesis in PsA patients and they suggest IL-7 involvement in the development of osteolysis in PsA.