INTRODUCTION

Progressive bone and cartilage destruction in arthritic joints leads to irreversible joint destruction, and subsequently to functional declines and work disability***(Pincus et al. 1984)***. New biomarkers such as cartilage oligometric matrix protein ***(Skoumal et al. 2004)***, osteoprotegerin ***(Schett, Redlich, and Amolen 2003),*** or receptor activator of NF-KB ligand ***(Herbier and Heufelder, 2001)*** have been developed to describe the local bone and cartilage process in affected joints.

Cathepsin K is a cysteine protease that plays an essential role in osteoclast function and in the degradation of protein components of bone matrix. It is produced by bone resorbing macrophages and synovial fibroblasts and it cleaves protein such as collagen type I, collagen type II and osteonectin ***(Hou et al. 2001).*** Cathepsin K therefore plays a role in bone remodeling and resorption in diseases such as osteoporosis, osteolytic bone metastasis and rheumatoid arthritis (RA) ***(Goto et al. 2003).***

Cathespin K is a tissue – specific protease associated with pycnodysotosis, a rare – genetic disorder that manifests itself in bone abnormalities such as short stature, acroosteolysis of distal phalanges and skull deformities ***(Singh and Singh, 2004).*** Inhibition of cathepsin K may therefore prevent bone resorption, as could be demonstrated in bone metastasis from breast cancer ***(Ishikawa et al., 2001).***

Osteoprotegerin has been shown to inhibit the expression of cathepsin K, the main enzyme involved in bone resorption.

AIM OF THE WORK

The aim of this study is to measure serum levels of cathepsin K in RA and to prove that cathepsin K is a parameter of bone destruction in a non selected cohort of patients with early RA.