

subsequent studies have shown that citrullination of  $\alpha$ -enolase is crucial for its autoantigenicity.  $\alpha$ -enolase is an evolutionary conserved protein implicated both in glycolysis pathway and as a plasminogen receptor. Here, the authors have evaluated, in the well-known collagen induced arthritis model, the clinical, immunological and histological effects of both recombinant non-citrullinated  $\alpha$ -enolase and immunodominant peptides from human and bacterial species.

**Methods** Different doses of  $\alpha$ -enolase (10 and 100  $\mu$ g) or immunodominant enolase peptide 1 from human (hEP1) or *porphyromonas Gingivalis* (pEP1) (10 or 100 $\mu$ g) were intraperitoneally injected to 6 week-old DBA/1 mice one day prior to collagen II arthritis induction (CIA). Both clinical (weight, arthritis score, tarsal thickness) and biological (anticollagen II and anti- $\alpha$ -enolase antibodies) were assessed during the 90 days follow-up period. Four histological score were also assessed: inflammation, synovial thickening, cartilage resorption and bone resorption.

**Results** Prophylactic injection of recombinant  $\alpha$ -enolase was able to significantly prevent weight loss and to decrease the severity of arthritis evaluated by the arthritis score as well as the tarsal thickness. There was a dose-effect since 100  $\mu$ g led to better results. Levels of anticollagen II antibodies were significantly lower whereas titers of anti- $\alpha$ -enolase antibodies were significantly higher in mice treated with 100  $\mu$ g of  $\alpha$ -enolase compared to control mice. Moreover, histological score were in agreement with clinical score. As regards to hEP1 and pEP1, the authors established a dose-dependant protective effect in CIA which is significant for pEP1. This protective effect is not due to once again a decrease of anti-collagen II antibodies titer.

**Conclusion** Prophylactic treatment with recombinant  $\alpha$ -enolase suggest a protective role of this molecule. The clinical effect is not due to an immunological response mediated by anti- $\alpha$ -enolase antibodies. Prophylactic injection could induce either an immune deviation or an emergence of regulatory lymphocyte population, responsible of a decrease of anti-CII antibodies production. These results suggest  $\alpha$ -enolase has an immunomodulatory effect in CIA mice. Those results suggest that non-citrullinated  $\alpha$ -enolase could constitute a potential new therapeutic approach in RA.

#### 4 PROPHYLACTIC INJECTION OF NON-CITRULLINATED $\alpha$ -ENOLASE HAS IMMUNOMODULATORY EFFECTS IN COLLAGEN-INDUCED ARTHRITIS MICE

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**Background** Identification of autoantibodies associated with rheumatoid arthritis (RA) has been of major interest. In this context, the authors have previously identified for the first time  $\alpha$ -enolase as a new auto-antigen in early RA. Moreover,