

EXPERIMENTAL RHEUMATOLOGY

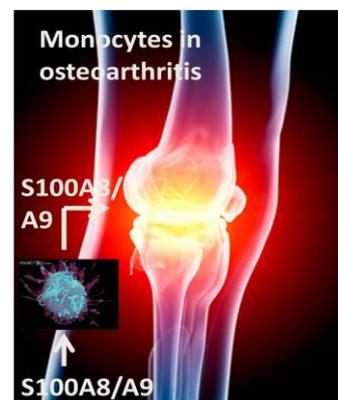
Balancing “alarming” monocytes suppresses development of joint pathology in osteoarthritis?

Clinical Relevance

Osteoarthritis (OA) is a joint disease that affects millions of people worldwide. It is one of the main causes of disability in the population, and is therefore a huge socioeconomic burden. Since the main risk factor for OA is aging, prevalence and incidence will further increase in the future. Present treatment consists of symptomatic treatment and eventually joint replacement surgery. A disease modifying treatment is not yet available. During OA the articular cartilage, which normally warrants smooth movement of the joints, is damaged and lost. The cause for this is not known, but it is now thought that it is not only a disease of the cartilage, but that also other tissues, like the synovium, are involved.

Background

In more than 50% of OA patients, the synovium is inflamed. During OA, monocytes from the blood migrate into the synovium. Recently a pro-inflammatory $Ly6C^{high}$ and an anti-inflammatory $Ly6C^{low}$ subpopulation has been described. Pro-inflammatory monocytes produce high amounts of S100 proteins. In previous studies we have found that alarmins S100A8/A9 are produced in high concentrations for prolonged periods and that they are crucial in mediating synovitis and joint pathology during experimental OA. High levels of S100A8/A9 might have effect on shifting monocytes towards a more pro-inflammatory subpopulation thereby aggravating joint destruction



Goals

Is S100A8/A9 able to shift monocytes towards a more pro-inflammatory population which remains in the OA joint and is responsible for the observed damage.

We Offer

You will work at a state of the art research laboratory which is renowned worldwide for its arthritis research and collaborate with a leading clinical rheumatology center, the St Maartenskliniek, both of which are combined in a EULAR Centre of Excellence in Rheumatology. You will work within a team that focuses on synovial activation and use techniques that include, but are not limited to, qPCR, imaging pro-inflammatory mediators, FACS analysis, cell culture, histology and immunohistochemistry. You will be able to improve your laboratory skills, develop your scientific thinking and expand your knowledge of underlying disease mechanisms. In addition you will improve your oral and written presentation skills.

Contact

Department: Experimental Rheumatology
Supervisor: Edwin Geven
Email address: Edwin.Geven@radboudumc.nl
Contact person: Peter van Lent
Telephone number: 31(0)24-3610512
Email address: peter.vanlent@radboudumc.nl
Website: www.experimentalrheumatology.nl



Radboudumc
university medical center