common between micro-organisms and human tissues are possible elements in autoimmunity.

THU0094 EXPERIMENTAL MYCOPLASMA FERMENTANS INFECTION IN RHEUMATOID SYNOVIAL FIBROBLASTS INDUCES M161AG EXPRESSION

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Background Mycoplasma fermentans (Mf) is one of the most likely of the several infectious agents which may be involved in the pathogenesis of rheumatoid arthritis (RA). Mf has been shown to induce arthritis experimentally in rabbits, and several reports have succeeded in detecting it in synovial fluids in RA patients. M161Ag is a 43-kDa surface lipoprotein of Mf, which induces cytokine production in monocytes and activates the host complement system.

Objectives The aim of this study was to investigate whether M161Ag can be induced on rheumatoid synovial fibroblasts by an Mf infected cell line *in vitro*.

Methods Rheumatoid synovial cells were isolated from synovial tissue specimens obtained at operation. Production of Mf infected rheumatoid synovial cells was performed using a coculture system of synovial cells and P39 (+) cells, which were Mf containing human leukaemia cell lines, for three days. PCR with mycoplasma specific primers were done to detect Mf DNA in synovial cells. M161Ag expression was evaluated by an immunohistochemical method with MK5 and MK53 monoclonal anti M161Ag antibodies, and mRNA production was also evaluated by the RT-PCR method.

Results Freshly isolated rheumatoid synovial cells were negative for Mf DNA using PCR with mycoplasma specific primers. DNA of Mf ribosomal RNA was detected in the synovial fibroblasts after coculture of synovial cells and P39 (+) cells by PCR with mycoplasma specific primers. Moreover, the expression of M161Ag was detected only on Mf infected synovial cells using the immunohistochemical method with MK5 or MK53 monoclonal antibodies.

Conclusion Experimental *in vitro* Mf infection in rheumatoid synovial fibroblasts was demonstrated to induce the expression of M161Ag. The significance of this finding requires clarification in further studies.

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THU0095 CIRCULATING AND LOCAL SYNOVIAL FLUID RENIN-ANGIOTENSIN SYSTEM IN PATIENTS WITH RHEUMATOID ARTHRITIS AND OSTEOARTHRITIS

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Background Recent researches demonstrated presence of local RAS in myocardium, ovary and brain. Angiotensin converting enzyme (ACE) was shown to be produced by synovial membrane macrophages. Angiotensin II is a potent vasoconstrictor, and also can stimulate angiogenesis, fibroblast proliferation and growth factor expression. Hypertrophy and hyperplasia of synovium and angiogenesis are well documented in persistent rheumatoid synovitis.

Objectives In this cross-sectional study we aimed to detect ACE and renin levels in serum and synovial fluid (SF) of patients with RA and osteoarthritis OA.

Methods Sixteen patients with RA (13F, 3M), 13 patients with OA (8F, 5M) and 11 healthy controls (HC) (5F, 6M) were enrolled in the study. The commercially available Angiotensin-Converting Enzyme kit and ACE calibrator were used. Renin assay was carried out with a two-site immunoreactive assay.

Results Median serum ACE levels were 48 U/l, 45 U/l, and 33 U/l in RA patients, OA patients, and HC, respectively. Median serum renin levels were 12.9 pg/ml, 11.8 pg/ml, and 17.9 pg/ml in RA patients and OA patients, respectively. Neither of these results were statistically significant ($p > 0.05$). SF levels of ACE and renin were found to be increased in RA patients (44 U/l and 3.5 pg/ml) compared to OA patients (24 U/l and 1.2 pg/ml) (Table 1). Synovial fluid renin levels in patients with RA was negatively correlated with disease duration.

Abstract THU0095 Table 1

	RA	OA	HControls
No of patients	16	13	11
Serum ACE (U/l)	48 (15–103)	45 (10–82)	33 (30–84)
Serum renin (pg/ml)	12.9 (1.2–67)	11.8 (1.6–39.2)	17.9 (17.9–36.6)
SF ACE (U/l)	44 (14–133)	24 (9–103)	
SF renin (pg/ml)	3.5 (0.48–15)	1.2 (0.5–4)	

Conclusion In this study we demonstrated that ACE and renin levels were elevated in SF of RA patients while serum ACE and renin levels were comparable to those in OA patients and healthy controls. These observations suggest presence of local RAS in RA synovium. Since angiotensin have stimulatory roles in angiogenesis and fibroblast proliferation, local RAS may participate in pannus formation and joint destruction in patients with RA. Negative correlation of synovial renin with disease duration may be compatible with previous observations that joint erosion is most prominent during the first months of the disease. Further studies, including mRNA of ACE and angiotensin II are needed to clarify exact roles of RAS in the pathogenesis of RA.

THU0096 EFFECT OF ANAKINRA (IL-1RA) AND SOLUBLE TUMOUR NECROSIS FACTOR RECEPTOR I (STNF-RI) ON CELLULAR IMMUNE FUNCTION IN RATS

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Background

Objectives This study investigated the potential for combined treatment with inhibitors of IL-1 and TNF- α to affect