50 ± 1 mg (dl, $p < 0.006$) by the treatment. The changes of the main parameters are summarised in Table 1.

Abstract THU0036 Table 1

	Before Remicade	After Remicade	Significant p-value
TC	209 ± 25	205 ± 36	
LDL-Chol	131 ± 24	118 ± 43	
HDL-Chol	56 ± 12	50 ± 13	$p < 0.006$
TG	112 ± 48	133 ± 53	$p < 0.01$
Lpa	1.1	1.4	

Values of the lipid parameters in our 15 patients.

Conclusion This study shows that elevated TG and decreased HDL levels occur in patients with RA and PsA after Infliximab therapy. We suggest that these changes in lipid metabolism may be a factor responsible for an increased development of vascular pathology.

THU0037 CLASS SPECIFIC RHEUMATOID FACTORS AND INTERLEUKIN-13 IN RHEUMATOID ARTHRITIS

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10.1136/annrheumdis-2001.834

Background In rheumatoid arthritis (RA) the production of rheumatoid factor (RF) by B-lymphocytes could depend on the action of different cytokines.

Objectives Aim of this study was to detect interleukin 13 (IL-13), a potent modulator of B-cell functions¹, in sera of RA patients in order to investigate the relationship of this cytokine, with different class specific RFs.

Methods We studied 36 consecutive patients with RA, classified according to Arnett criteria and 20 healthy subjects matched for age and sex. In all patients, we evaluated the main clinical and laboratory parameters including the determination of IgM-RF, IgA-RF, IgG-RF and IL-13 by immunoenzymatic methods.

Results We found detectable levels of IL-13 (>1.56 pg/ml) in 86.1% of RA patients and in 40% of controls ($p < 0.0006$). The IL-13 serum levels were significantly ($p < 0.00001$) higher in patients with RA (mean/lower and upper 95% CI = 165.4/99.3–231.6 pg/ml) respect to the controls (mean/lower and upper 95% CI = 3.9/0.8–7.0 pg/ml). The serum levels of IL-13 correlated with those of IgM-RF ($r = 0.591$; $p < 0.0001$) and IgA-RF ($r = 0.505$; $p < 0.002$), while we didn't find any relationship with IgG-RF, IgM, IgA and IgG levels. IgA-RF levels correlated with those of IgM-RF ($r = 0.548$; $p < 0.0005$) and IgG-RF ($r = 0.543$; $p < 0.0006$), while no relationship between IgM-RF and IgG-RF ($r = 0.222$; n.s.) was found. The comparison of patients with or without the different class specific RFs revealed that patients with IgM-RF had significantly ($p < 0.018$) higher levels of IL-13 respect to the patients without (mean/lower and upper 95% CI = 202.4/125.8–278.9 vs 12.6/2.2–22.9 pg/ml), while this finding was not observed for IgA-RF (mean/lower and upper 95% CI = 191.1/114.3–267.9 vs 59.3/58.6–177.2 pg/ml) and IgG-RF (mean/lower and upper 95% CI = 239.4/48.6–430.2 vs 147.6/74.5–220.7 pg/ml). We didn't find any relationship of serum IL-13 levels with clinical and other laboratory signs of disease activity.

Conclusion In conclusion our results show that IL-13 is involved in pathogenetic mechanism of RA, suggesting that IL-13 source could derive from an excessive joint or intravascular production by immune system. A relevant action of IL-13 seems to be related with a selective isotype production of IgM-RF.

REFERENCE

- 1 Punnonen J, Aversa G, Cocks BG, McKenzie ANJ, Menon S, Zurawski G, De Waal Malefyt R, De Vries JE. Interleukin 13 induces interleukin-4-independent IgG4 and IgE synthesis and CD23 expression by human B cells. *Proc Natl Acad Sci USA* 1993;**90**:3730–4

THU0038 CYTOKINE AND GROWTH FACTOR REGULATION OF RA AND OA SYNOVIAL MACROPHAGE DIFFERENTIATION INTO OSTEOCLASTS AND DENDRITIC CELLS

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10.1136/annrheumdis-2001.835

Background Rheumatoid arthritis (RA) is a chronic autoimmune inflammatory disease in which an unknown autoantigen is presented to T cells. In RA synovial tissue, fully differentiated dendritic cells (DC) are found in close association with T cells. DC are thought to be the major antigen presenting cells in RA. It has previously been shown that human monocytes differentiate into DC when stimulated with GM-CSF and IL-4 with terminal differentiation promoted by TNF α . We have shown that circulating monocytes and synovial macrophages are stimulated to differentiate into functional osteoclasts in the presence of RANKL, M-CSF and dexamethasone (Dex).

Objectives We aimed to determine whether synovial macrophages preferentially differentiate into DC or functional osteoclasts in the presence of specific combinations of cytokines/growth factors which are known to be involved in the pathogenesis of RA and other inflammatory bone conditions.

Methods Synovial macrophages were isolated from the knee and hip of RA and OA patients. The cells were seeded onto dentine slices and coverslips and cultured for 1, 7 and 14 days in the presence/absence of different combinations of the following factors: RANKL (30 ng/ml), Dex (10–8M), MCSF (0–50 ng/ml), GM-CSF (0–50 ng/ml), IL-4 (10 ng/ml), TNF α (10 ng/ml). Macrophage-osteoclast differentiation was quantified by counting the number of multinucleated, tartrate-resistant acid phosphatase (TRAP) cells present, and by measuring their ability to carry out lacunar resorption on dentine slices. Mature DC were identified by the expression of the marker CD83.

Results In the presence of RANKL, M-CSF and Dex, RA and OA synovial macrophages were maximally stimulated to form osteoclasts; a few cells in these cultures were CD83 positive. The majority were TRAP positive and capable of forming resorption pits on dentine. GM-CSF, IL-4 and TNF α stimulated maximum expression of the mature DC marker CD83 in the cultures; these cultures were TRAP negative and incapable of resorbing dentine. Synovial macrophages cultured in the presence of RANKL and GM-CSF alone resulted in the formation of large multinucleated cells which expressed the marker CD83; a few of these large multinucleated cells were also TRAP positive. Cell cultures treated with GM-CSF were capable of significantly less dentine resorption than cultures treated with RANKL and M-CSF. The addition of RANKL did not modify CD83 expression in any of the above cytokine/growth factor combinations.