Sirs,

Reactive arthritis (ReA) is triggered by urethritis or enteritis causing pathogens (1). The presence of IL-17 has been reported in ReA joints following a history of diarrhea (2) but the presence of IL-17 in C. trachomatis ReA joints has never been investigated. The objective of the study described herein was to determine the levels of IL-17 and IL-17-modulating cytokines in the synovial fluid (SF) from C. trachomatis ReA, rheumatoid arthritis (RA) taken as inflammatory control, and osteoarthritis (OA) as non-inflammatory control patients:

(i) C. trachomatis ReA (n=13) was defined by the presence of asymmetrical mono/oligoarthritis and evidence of C. trachomatis infection (positive culture or DNA amplification) (median age, 26 years; interquartile range (IQR) 21-33, 2 were females, median number of SF leukocytes, 13700/mm³ (9400–23900), median duration of arthritis, 14 days (IQR 7–46) and median number of active joints, 2 (IQR 1–3.3)).

(ii) Rheumatoid arthritis (RA) (n=20) (median age, 67 years (IQR 54–79), 17 females, median number of SF leukocytes, 7700/mm³ (IQR 5738–12613)).

(iii) Osteoarthritis (OA) (n=17) (median age, 73 years (IQR 64–78), 11 females, median number of SF leukocytes, 300/mm³ (IQR 150-463)).

The levels of cytokine concentrations in SF were determined by sandwich ELISA techniques. In C. trachomatis ReA patients, the SF concentrations of IFN-γ and IL-17 were significantly higher than in OA patients (median 13.6 pg/ml, IQR 2.33–31.3 vs median 0 pg/ml, IQR 0–1.76 p<0.005 and median 5.7 pg/ml, IQR 0–25.4 vs median 0 pg/ml, IQR 0–0, p<0.005, respectively). No significant difference was found between C. trachomatis ReA and RA patients (Fig. 1).

A positive correlation (Rho=0.74, p=0.011) was found between the levels of IFN-γ and IL-17 in SF of C. trachomatis ReA patients. The levels of cytokines modulating IFN-γ and IL-17 expression such as IL-1β, IL-6, IL-12p70 and IL-23(p19/p40), (3, 4) were also determined. The concentrations of IL-1β in the SF of C. trachomatis ReA (median 6.5 pg/ml, IQR 1.6–15.2) were significantly higher than in OA patients (median 0 pg/ml, IQR 0.1, p<0.0001) (Fig. 1). A positive correlation (Rho=0.70, p=0.02) was found between the levels of IL-1β and IL-17 in SF of C. trachomatis ReA patients. There was no significant difference between ReA and OA regarding the other cytokines, mainly because of wide interindividual variations.

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**Fig. 1.** Levels of IFN-γ, IL-17, IL-6, and IL-1β in the SF of patients with C. trachomatis ReA, RA and OA. Levels were determined by ELISA. Horizontal bars within boxes show the median, boxes show the interquartile range and vertical bars show the 95% confidence interval (values above and below these levels were plotted separately). Comparison between two groups was made only when the Kruskal-Wallis test yielded statistically significant results.

**p<0.005; ***p<0.0001 compared with OA patients (Mann-Whitney test).**
in IL-6 concentrations and non detectable levels of IL-12p70 and IL-23 (data not shown). This is the first study reporting the presence of IL-17 in the SF of patients with C. trachomatis ReA. Both IFN-γ and IL-17 were detected and a positive correlation was found between their levels indicating possible similar regulation by the local cytokine milieu in C. trachomatis ReA joints. Both IFN-γ and IL-17 were detected and a positive correlation was found between their levels indicating possible similar regulation by the local cytokine milieu in C. trachomatis ReA joints.

The role of IFN-γ and IL-17 in the SF from C. trachomatis ReA patients is unclear. IFN-γ is well known for its antichlamydial activity but its median concentration (13.6 pg/ml) was lower than values reported to be efficient in vitro. Indeed, high levels of IFN-γ (2 ng/ml) promoted the destruction of Chlamydia whereas lower levels (0.2 ng/ml) induced the formation of persistent forms (5). However it is difficult to extrapolate results obtained in vitro to the environment of an arthritic joint. The same remark is valid for IL-17 concentrations found in SF from C. trachomatis ReA patients. IL-17 is well known for its role in joint destruction (6) but recent findings indicate that IL-17 may also contribute to protection against intracellular bacteria (7, 8).

References