suggest that FN might play a protective role by a reduction of control group. Thus, the absence of CX3CL1-CXCR1 interaction in kUCed enteritis to 5 days after cisplatin challenge in IFN-γ mice. These observations indicated that CX3CL1-CXCR1 signal pathway could play a protective role in C. difficile toxin A-induced enteritis.

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A novel classification of collagen diseases utilizing Th1/Th2/Th17 cytokine profile in the peripheral blood mononuclear cells and change of the profile before and after infliximab therapy: MIYOSHI Fumihiko, SATO Kojiro, MIMURA Toshihide (Division of Rheumatology and Applied Immunology, Saitama Medical University, Japan) Background: RA and SLE have been considered a Th1- and a Th2-type disease, respectively. INF-γ and IL-2, the key cytokines produced from Th1 cells, however, were barely detected in the affected joints of RA patients. This result suggests that RA is not in fact a Th1 disease. Recently, Th17 has been identified as an important player in inflammatory responses. Several studies have suggested that IL-17 plays an essential role in the pathogenesis of some autoimmune diseases including RA. Objectives: The aims of the present study were to re-classify collagen diseases by taking Th17 subset into consideration and to understand how Th1/17 subsets interact each other in the process of disease development. Methods: We obtained mononuclear cells from the PBMC of patients with various collagen diseases (RA, SLE and Behcet's disease) and normal controls. The cells were stimulated with PMA/ionomycin in vitro for 5 hours. The supernatant of the culture and total RNA from the cells were collected. The expression of INF-γ, IL-4 and IL-17 at both mRNA and protein levels were measured. Results: Unique patterns were observed in terms of the Th1/17 ratios, depending on the type of the disease. For example, a Th1- and a Th17-type response were dominant in Behcet's disease patients. SLE patients also showed a Th17-type response rather than a Th1-type response. RA patients did not show an evident skewing in Th1/17 balance. Surprisingly, the expression levels of the cytokines before the infliximab treatment were lower than those of normal controls and they became higher and came closer to normal after the therapy. We are now trying to interpret these unexpected results by employing mouse CIA. Our next step will be to construct a simulation model that can explain both human clinical data and mouse experimental data.