antigen was detected as long as three and 17 years after yersinia infection, suggesting that the microbial structures can persist in the tissues. The antigens are found in mesenteric and cervical lymph nodes and in the skin, and, during the acute phase of infection, in peripheral blood cells of almost every patient, including patients who will not develop reactive arthritis. Thus the presence of antigens in the blood provides an explanation for extensive antigen dissemination, but not for the development of reactive arthritis and other clinically significant extraintestinal manifestations.

Possibly, extraintestinal symptoms derive from inflammatory hyperreactivity of the patient to the antigenic stimulus. It has been reported that neutrophils of HLA-B27 positive subjects without a history of yersinia infection and of patients with previous yersinia reactive arthritis show enhanced neutrophil migration in response to a chemotactic stimulus in vitro and in vivo, as do patients with ankylosing spondylitis, at least in vitro. Furthermore, neutrophils from patients who have a history of severe acute yersinia triggered reactive arthritis, or with sequelae, show increased generation of oxygen radicals in vitro. NAP-1/IL-8 stimulates neutrophil chemotaxis and oxygen radical production and might thereby contribute to the neutrophil hyperactivity.

In conclusion, the results show that both control and lipopolysaccharide induced NAP-1/IL-8 production by monocytes was similar in subjects with past yersinia arthritis or enteritis and unaffected subjects, and did not differ from HLA-B27 positive and negative subjects. This seems to rule out an aberrant function of monocytes, at least for the synthesis and release of NAP-1/IL-8, one of their major products, in the triggering of reactive arthritis.

We thank Heidi Mühlschläger, Paula Rahikainen, and Eire Virstamäki for technical help. The study was supported by grants from the Yrjö Jahnsson Foundation, Helsinki, and the Finnish Cultural Fund, Helsinki, Finland.