

- 10 Vuorinen-Markkola H, Yki-Järvinen H. Hyperuricemia and insulin resistance. *J Clin Endocrinol Metab* 1994; **78**: 25-9.
- 11 Facchini F, Chen I, Hollenbeck CB, Reaven GM. Relationship between resistance to insulin-mediated glucose uptake, urinary uric acid clearance, and plasma uric acid concentration. *JAMA* 1991; **266**: 3008-11.

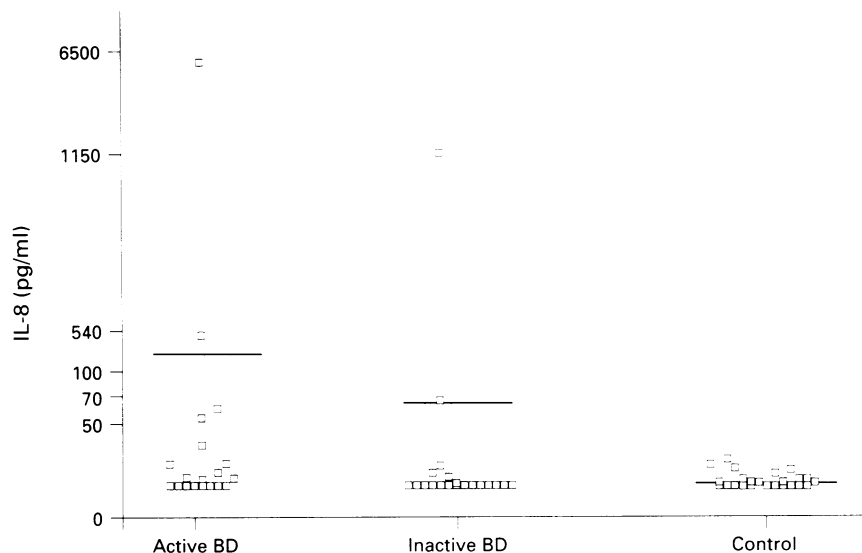
Serum levels of interleukin-8 in patients with Behçet's disease

Interleukin-8 (IL-8) or neutrophil activating peptide-1 (NAP-1) is produced by a variety of cell types including endothelial cells, synovial cells, keratinocytes, fibroblasts, and monocytes-macrophages.¹⁻⁴ IL-8 has been shown to induce neutrophil degranulation and to be a potent chemoattractant for neutrophils.^{2,5} Furthermore, IL-8 activated neutrophils are a major source of the enzymes involved in tissue destruction.⁵

Behçet's disease (BD) is an inflammatory vasculitis of unknown cause characterised by orogenital ulceration and variably associated with uveitis, arthritis, and skin lesions.⁶ In addition to the migration and activation of mononuclear cells, a neutrophilic vascular reaction was reported to be the fundamental histological change in certain lesions of BD.^{7,8} For this reason, we studied the serum levels of IL-8 in patients with BD and healthy controls, and the possible relationship with disease activity in BD.

Forty patients with BD, all fulfilling the International Study Group criteria for the diagnosis of BD⁹ and 25 apparently healthy controls were studied. At the time of the blood withdrawal, 20 patients (12 men, eight women; mean age 36.4 (SD 7) years, range 25-54) having at least two of the following were considered as having active disease: oral ulcer, genital ulcer, eye lesions, skin lesions, arthritis, pulmonary involvement and vascular lesions (venous thrombosis, arterial aneurysm) and 20 patients (14 men, six women; mean age 37.5 (8.6) years, range 22-54) showing no clinical or laboratory disorder related with BD for at least one month were considered as having inactive disease. All but three patients with active disease were receiving colchicine 1-1.5 mg/day. Patients with inactive disease were receiving low-dose colchicine (0.5-1.0 mg/day). Sera were stored at -70°C until required for use.

Serum IL-8 was determined by Biotrak Interleukin-8, human enzyme linked immunosorbent assay system kit (Amersham), which uses the quantitative immunometric, sandwich enzyme immunoassay technique. All samples were tested in duplicate. Intra-assay and inter-assay variation for this kit were 7.5% and 9.1%, respectively, and the lower limit of detection of IL-8 was 18.1 pg/ml.



Serum interleukin-8 (IL-8) levels in patients with Behçet's disease (BD) and healthy controls.

Erythrocyte sedimentation rate (ESR) was determined by the Westergren method and C reactive protein (CRP) by nephelometry (Behring). The IL-8 data were logarithmically transformed before statistical analysis and groups were compared by Student's *t* test and correlation analysis.

The mean (SD) IL-8 concentration was 369.3 (1447.8) pg/ml in patients with active BD, 65.6 (255.8) pg/ml in patients with inactive disease, and 13.1 (12.3) pg/ml in healthy controls (figure). Although the mean IL-8 values in active and inactive BD appeared higher than those in the control group, the difference was not statistically significant ($p > 0.05$). Mean serum IL-8 levels of patients with active BD were greater than those of patients with inactive BD, but the difference was again statistically insignificant ($p > 0.05$). Clinical disease activity correlated well with both ESR and CRP ($r = 0.665$, $p < 0.001$), but there was no correlation of IL-8 with ESR or CRP ($p > 0.05$).

In conclusion, the serum level of IL-8 in BD patients was not significantly different from that in healthy controls. Further studies are needed to define the specific function of this cytokine in BD, and IL-8 studies in tissue samples may provide useful information concerning the aetiopathogenesis of BD.

KÜRSAT ÖZORAN
OLCAY AYDINTUĞ
GÜNER TOKGÖZ
NURŞEN DÜZGÜN
HÜSEYİN TUTKAK

Departments of Immunology-Rheumatology,
Faculty of Medicine, Ankara University,
Ankara, Turkey

AYSEL GÜRLER
Department of Dermatology

- 1 Thornton A J, Strieter R, Lindley I, Baggolini M, Kunkel S L. Cytokine induced gene expression of neutrophil chemotactic factor/interleukin-8 in human hepatocytes. *J Immunol* 1990; **144**: 2600-13.
- 2 Matsumoto T, Miike T, Nelson R P, Trudeau W L, Lockey R F, Yodoi J. Elevated serum levels of IL-8 in patients with HIV infection. *Clin Exp Immunol* 1993; **93**: 149-51.
- 3 Deleuran B, Lemche P, Kristensen M, et al. Localisation of IL-8 in the sinovial membrane, cartilage-pannus junction and chondrocytes in rheumatoid arthritis. *Scand J Rheumatol* 1994; **23**: 2-7.
- 4 Brennan F M, Zachariae C O C, Chantry D, et al. Detection of interleukin 8 biological activity in synovial fluids from patients with rheumatoid arthritis and production of interleukin 8 mRNA by isolated synovial cells. *Eur J Immunol* 1990; **20**: 2141-4.
- 5 Peichl P, Ceska M, Broell H, Effenberger F, Lindley I J D. Human neutrophil activating peptide/interleukin 8 acts as an autoantigen in rheumatoid arthritis. *Ann Rheum Dis* 1992; **51**: 19-22.
- 6 Hamzaoui K, Ayed K, Slim A, Hamza M, Touraine J. Natural killer cell activity, interferon gamma and antibodies to herpes viruses in patients with Behçet's disease. *Clin Exp Immunol* 1990; **79**: 28-34.
- 7 Gilhar A, Wintwrstein G, Turani H, Landau J, Etzioni A. Skin hyperactivity response (pathergy) in Behçet's disease. *J Am Acad Dermatol* 1989; **21**: 547-52.
- 8 Helm T N, Camisa C, Allen C, Lowder C. Clinical features of Behçet's disease. *Oral Surg Oral Med Oral Pathol* 1991; **72**: 30-4.
- 9 International Study Group (ISG) for Behçet's Disease. Evaluation of diagnostic criteria in Behçet's Disease. *Br J Rheumatol* 1992; **31**: 299-308.