MLC concentrations will be useful in the assessment of such a response remains to be determined. A previous preliminary report revealed high MLC levels in 75% of patients with myositis,[8] but a correlation with disease activity was not performed. Further prospective longitudinal studies are in progress to determine the utility of measuring MLC concentrations in myositis.

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Decreased triglyceride levels with low calorie diet and increased renal excretion of uric acid in hyperuricaemic-hyperlipidaemic patients

While the association between hyperuricaemia and hyperlipidaemia is well known, it has so far been poorly explained.[1] The presence of external factors such as obesity, increased alcohol intake,[2] or some nutritional habits[3] in the same individual was initially believed to cause both metabolic disturbances. However, this has been shown not to be the case in many instances; hyperuricaemia and hyperlipidaemia have been observed in the absence of those factors,[4] and even been ascribed to a common genetic basis.[5] In addition, hyperuricaemic-hyperlipidaemic patients have been shown to exhibit decreased renal excretion of urate relative to hyperuricaemic-normolipidaemic individuals.[6]

Metabolic parameters and renal excretion of urates for the two patient groups before (A) and after (B) low calorie dieting

	Group I A	*Group II A*	*Group I B*	*Group II B*
Body mass index	30·1 (3·5)	29·8 (3·6)	28·7 (3·4)***	28·2 (3·2)**
Serum uric acid (mg/dl)	8·3 (1·6)	8·4 (1·6)	8·0 (1·6)	7·5 (1·5)
Total cholesterol (mg/dl)	192 (24·2)	261·3 (40·9)††	173·5 (33·6)*	204·1 (41·0)***†
Total triglycerides (mg/dl)	133 (44·8)	278 (53·3)†††	90·3 (42·5)**	161·5 (117)***†
Uric acid excretion (mg/24 h)	621 (215)	605 (194)	629 (347)	715 (157)*
Fractional excretion uric acid (%)	5·8 (1·4)	6·0 (2·5)	6·2 (2·5)	8·2 (3·2)*†

Values are mean (SD). Group I = primary hyperuricaemic; group II = primary hyperuricaemic-hyperlipidaemic. Significant differences: *p < 0·05, **p < 0·01, ***p < 0·001 for comparison of A with B; †p < 0·05, ††p < 0·01, †††p < 0·001 for comparison of group I with group II.

In order to investigate alterations in the renal excretion of uric acid in relation to plasma concentrations of triglycerides, we carried out a dietary intervention study in hyperuricaemic patients who were placed on a controlled low calorie regimen intended to decrease their triglyceride concentrations.

We studied 15 primary hyperuricaemic patients (all men) (group I) and 15 primary hyperuricaemic-hyperlipidaemic patients (all men) (group II). Subjects with plasma uric acid concentrations greater than 7 mg/dl were classed as hyperuricaemic, and those with values greater than 200 mg/dl,[7] as hyperlipidaemic. All were subjected to an initial basal analytical determination for uric acid, total triglycerides, total cholesterol, and uric acid clearance and fractional excretion after three days on a low purine diet,[8] followed by a second determination after three weeks on a low calorie diet (1200 kcal per day: carbohydrate = 50%; protein = 20%; lipid = 30%) with alcohol excluded. Three days after the second determination, patients were again placed on a low purine diet similar to that used before the first determination.

The Wilcoxon test was used to evaluate the significance of differences between the means in the two patient groups before and after low calorie dieting. Statistically significant difference between the two groups was calculated by the Mann-Whitney test.

The table shows the results. All the patients lost a significant amount of body weight on dieting, regardless of the group to which they belonged, but uric acid levels did not change in either group as a result of dieting.

Patients in group II (hyperuricaemic-hyperlipidaemic) exhibited considerably decreased triglyceride and cholesterol concentrations after the low calorie diets, concomitant with increased renal excretion of uric acid, which was not observed in group I.

Our results support the hypothesis that the association between triglyceride concentration and renal excretion of uric acid is more than a casual relationship; by decreasing triglyceride concentrations, we succeeded in increasing renal uric acid excretion without pharmacological intervention, while controlling the purine intake during assessment of the renal excretion of uric acid. In our opinion, the relationship is particularly valid in hyperuricaemic-hyperlipidaemic patients, as the decrease in the triglyceride concentrations and weights of the hyperuricaemic-normolipidaemic men (group I) was not accompanied by increased renal excretion of uric acid. Collantes *et al*[6] also found that renal excretion of urate was less in hyperuricaemic-hyperlipidaemic patients than in hyperuricaemic-normolipidaemic patients. Acute increase in serum triglyceride concentrations has been shown not to modify uric acid synthesis or excretion.[3 9] However, these data are not conclusive, as ingested triglycerides have a different composition and metabolic origin than endogenous triglycerides.

Other authors have shown an inverse correlation between insulin sensitivity and uric acid concentration,[10] and between insulin resistance and clearance of uric acid from healthy volunteers.[11] The potential link between the inverse relationship of the renal excretion of uric acid with hyperinsulinism and our findings remains to be determined. The mechanisms responsible for such an inverse relationship between renal excretion of uric acid and triglyceride concentrations will probably be elucidated as knowledge of the metabolic syndrome expands.

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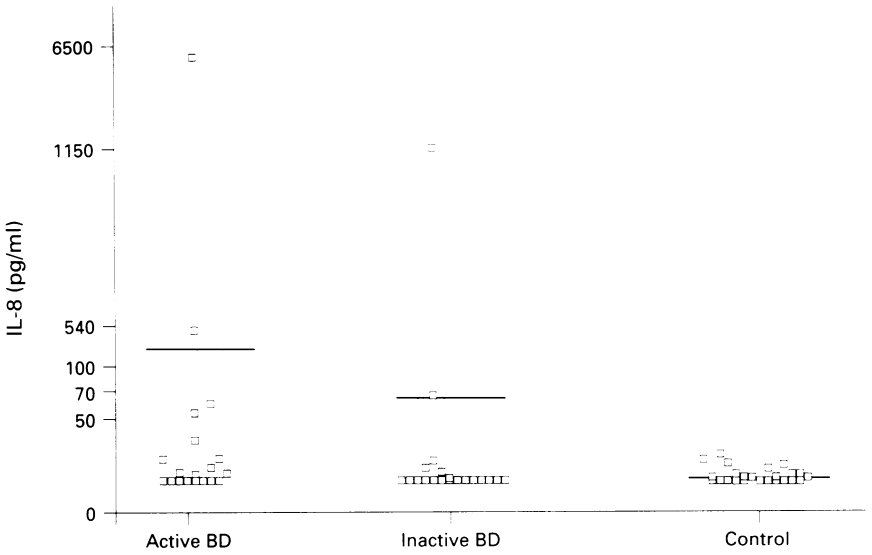
Serum levels of interleukin-8 in patients with Behçet's disease

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 BD[9] 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 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.

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