Dear Sir,

In their recent report, Zlatkov et al. [1] demonstrated serum antibodies to DNA in psoriatics in about 20%. Antibodies to single-stranded DNA had been detected in psoriatic sera with or without photo(che-mo)therapy by Meffert et al. [2, 3] using the Farr technique too. Interestingly, they observed a slight decrease after selective UV therapy (SUP). Intensive sunlight exposure failed to produce significantly raised anti-DNA levels [1].

Using a fluorescence enzyme-linked immuno-sorbent assay for antibodies to double-stranded DNA [4] we detected borderline concentrations in psoriatic sera [5]. On average, the antibody concentration in psoriatic arthritis was higher than in psoriasis vulgaris. The findings are supported by others [2, 3]. As demonstrated by Zlatkov et al. [1] and others [2, 3], anti-DNA and the clinical course are likely to be independent.

A chronic inflammatory DNA liberation associated with T cell imbalance might be responsible for the antibody production. It supports the hypothesis of an immune or quasi-immune handling of native and denatured DNA as a physiological mode of action [6]. Since therapeutic management leads only to a nonsignificant variation of the anti-DNA concentration, these antibodies are not to be candidates in the underlying pathological process.

References