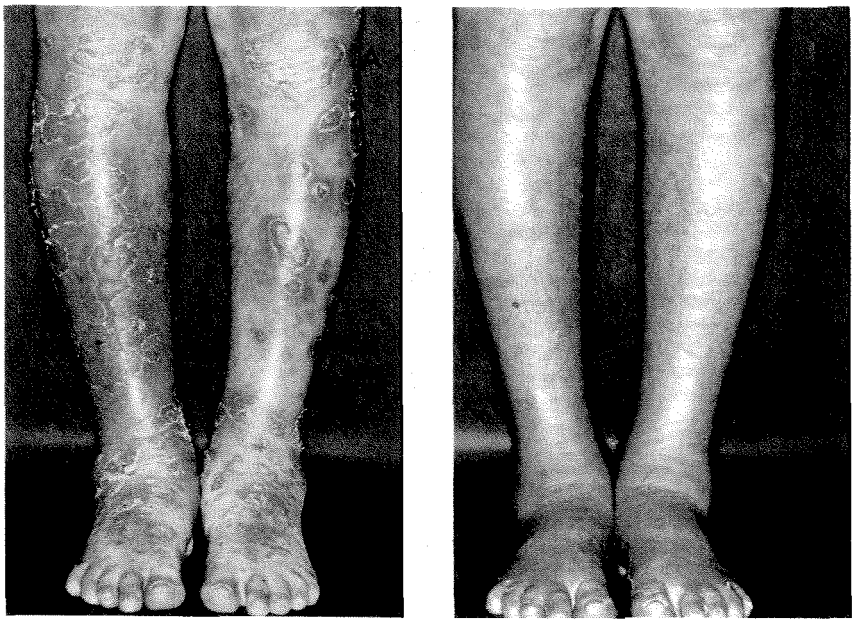

Appearance of legs (A) before and (B) 11 days after first CD4 antibody infusion.

role in this disorder. Not only the generalised exacerbation of psoriasis but also the chronic psoriatic plaques responded. The nature of the putative, epidermis-derived, stimulus-inducing T-cell activation and infiltration remains unknown.

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CD4 antibody treatment of severe psoriasis

SIR,—Dr Poizot-Martin and his colleagues (June 15, p 1477) suggest that CD4 monoclonal antibodies and peptide T could be helpful in the treatment of resistant psoriasis. We have initiated a phase II clinical trial of a CD4 monoclonal antibody in generalised severe psoriasis and we report this treatment in three patients.

The first patient, a 61-year-old man who had psoriasis vulgaris for 23 years, had a severe psoriatic erythroderma with a psoriasis area sensitivity index (PASI) of 35 a few days before entering the study. He received daily 2 h infusions of CD4 antibody (clone BB14, murine IgG_1, 0·2 mg/kg per day) for 8 days. Clinical tolerance was very good, apart from chills during the first infusion. Improvement started on the fourth day of treatment and was greatest at day 30 (PASI, 12). He deteriorated progressively within a 2-month follow-up (PASI, 20).

The two other patients (aged 40 and 32), with disease duration of 10 and 13 years, respectively, had chronic psoriasis (PASI, 15 and 16). Previous treatments, including retinoids and methotrexate, had been withdrawn because of lack of efficacy and/or toxic side-effects. These patients received CD4 antibody (0·8 mg/kg per day for 3 days, then 0·4 mg/kg per day for 5 days). Clinical improvement was observed on day 8 (PASI, 10 and 8, respectively) and was greatest after 3–4 weeks (PASI, 0 and 4, respectively). In all three patients histological examination of healed skin lesions taken at day 30 showed an almost normal appearance of the skin architecture, with some signs of fibrosis but without any epidermal or dermal signs of psoriasis.

The pathogenesis of psoriasis is still unclear but recent studies have emphasised the role of activated CD4 cells infiltrating the lesional skin.[1,2] The therapeutic activity of cyclosporin[2] and our results with CD4 monoclonal antibodies support this hypothesis and suggest that CD4 cells may be relevant targets for immunointervention in the most severe forms of this disease.

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Detection of *Chlamydia pneumoniae*

SIR,—We were surprised by the information put forward by Dr Hahn and Ms Dodge (April 6, p 849) to indicate the sensitivity and specificity of enzyme immunoassay (EIA) and direct fluorescent antibody (DFA) techniques for detecting *Chlamydia pneumoniae*. They diluted a suspension of *C pneumoniae* and inoculated each dilution onto McCoy cell monolayers (in triplicate). After incubation, the number of inclusions in one set of monolayers was assessed; the second set of monolayers was swabbed and the swabs tested by EIA; and the third set was swabbed and the swabs tested by DFA. However, since the number of elementary bodies on the swab available for testing by EIA or DFA after swabbing a monolayer may bear no relation to the number of inclusions counted in the monolayer, and certainly have no relation to the number of elementary bodies expected at a particular dilution of the