

Serum IgE before and after Treatment for Scabies

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In 120 patients, treated for scabies infection, the serum IgE concentrations fell from high levels in 54 and normal levels in 66 to lower levels 12 months after treatment. The specific IgE antibodies to *Dermatophagoides pteronyssinus* (RAST) were also reduced 12 months after treatment. The decrease in both IgE concentrations and IgE antibodies was most conspicuous in non-atopic patients. These findings confirm previous observations of specific immunological hypersensitivity to scabies mite, and that scabies infection stimulates the production of IgE antibodies.

Key words: scabies; IgE stimulation; specific immunological hypersensitivity.

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Elevated serum immunoglobulin concentrations, especially IgE, have been reported in many intestinal or systemic parasitic diseases caused by helminths, as for example ascariasis (7), visceral larvae migrans (6), capillariasis (10), and trichinosis (11), whereas protozoan infections such as giardiasis, amoebiasis, trypanosomiasis and malaria do not elevate the serum IgE levels (4).

Based upon clinical and epidemiological manifestations there is evidence that the immune system is involved in the pathogenesis of scabies (5, 8). Serum immunoglobulin concentrations, especially IgE concentrations, are often altered during scabies infection (1). In a high proportion of patients with scabies, IgE antibodies to the house dust mite *Dermatophagoides pteronyssinus* (DP) can be demonstrated (2). Specific immunological hypersensitivity to scabies mite has been demonstrated by *in vivo* experiments (3). Since the house dust mite and the human scabies mite are arthropods of similar appearance, and with similar nutrition, it is not unlikely that they or their excretions or secretions have allergens in common. In view of this hypothesis, specific IgE antibodies to house dust mites were measured in patients with scabies.

The present report concerns follow-up studies of serum IgE concentrations and antibodies to DP after treatment for scabies.

MATERIAL AND METHODS

Individuals

Of 120 persons with scabies, previously reported (2), 40 had a positive (Fig. 1 A and 1 C), and 80 had a negative (Fig. 1 B and 1 D) RAST to DP. Of the 40 persons with a positive RAST to DP, 34 had elevated serum IgE concentrations, and of the 80 persons with a negative RAST to DP, 20 had elevated serum IgE concentrations.

Based upon interviews, direct skin tests (prick test) and the finding of specific circulating IgE antibodies other than DP, atopic disease was highly probable in 40 of the 120 persons (Fig. 1 C and 1 D), of which 22 had a positive (Fig. 1 C), and 18 a negative (Fig. 1 D) RAST to DP. Of the 40 atopic persons 24 had elevated serum IgE concentrations, and of the 80 non-atopic persons 30 had elevated serum IgE concentrations. Atopic patients are here defined as patients with either current atopic disease or a past personal history of such illness, including atopic dermati-

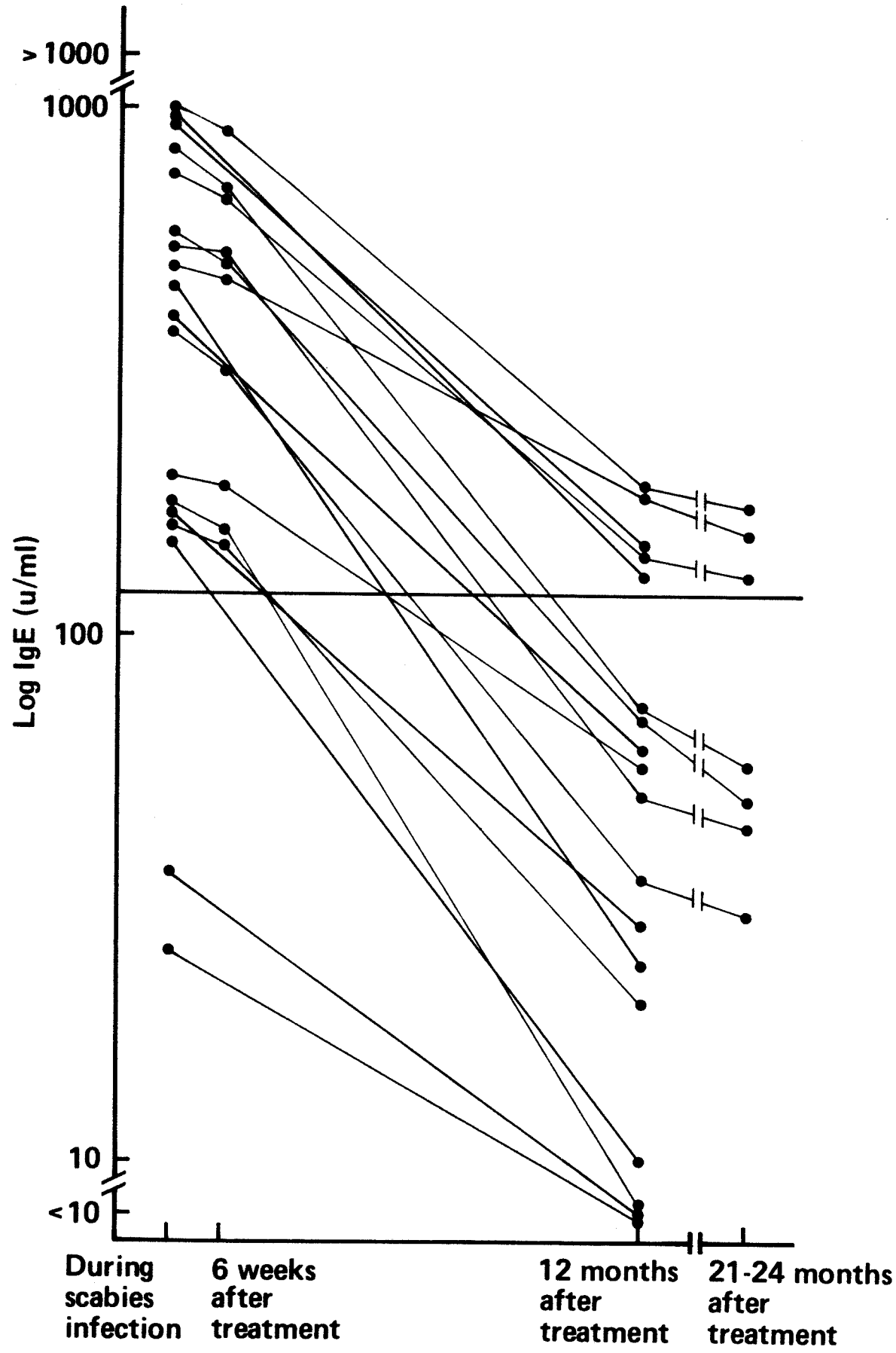


Fig. 1 A. IgE concentrations in 18 non-atopic patients with a positive RAST to *D. pteronyssinus* during scabies infection, 6 weeks, 12 months, and 21-24 months after successful treatment.

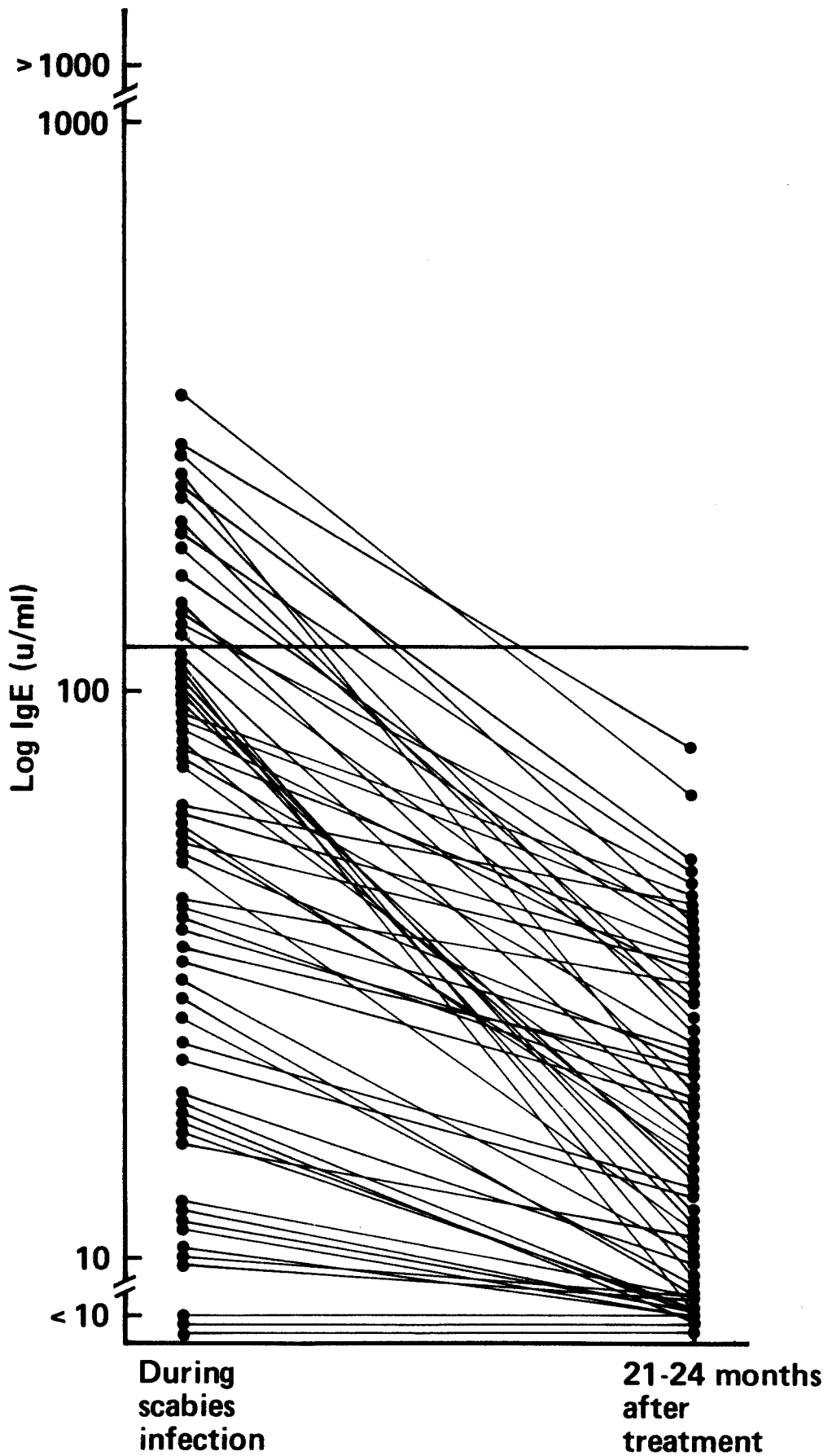


Fig. 1 B. IgE concentrations in 62 non-atopic patients with a negative RAST to *D. pteronyssinus* during scabies infection and 12 months after successful treatment.

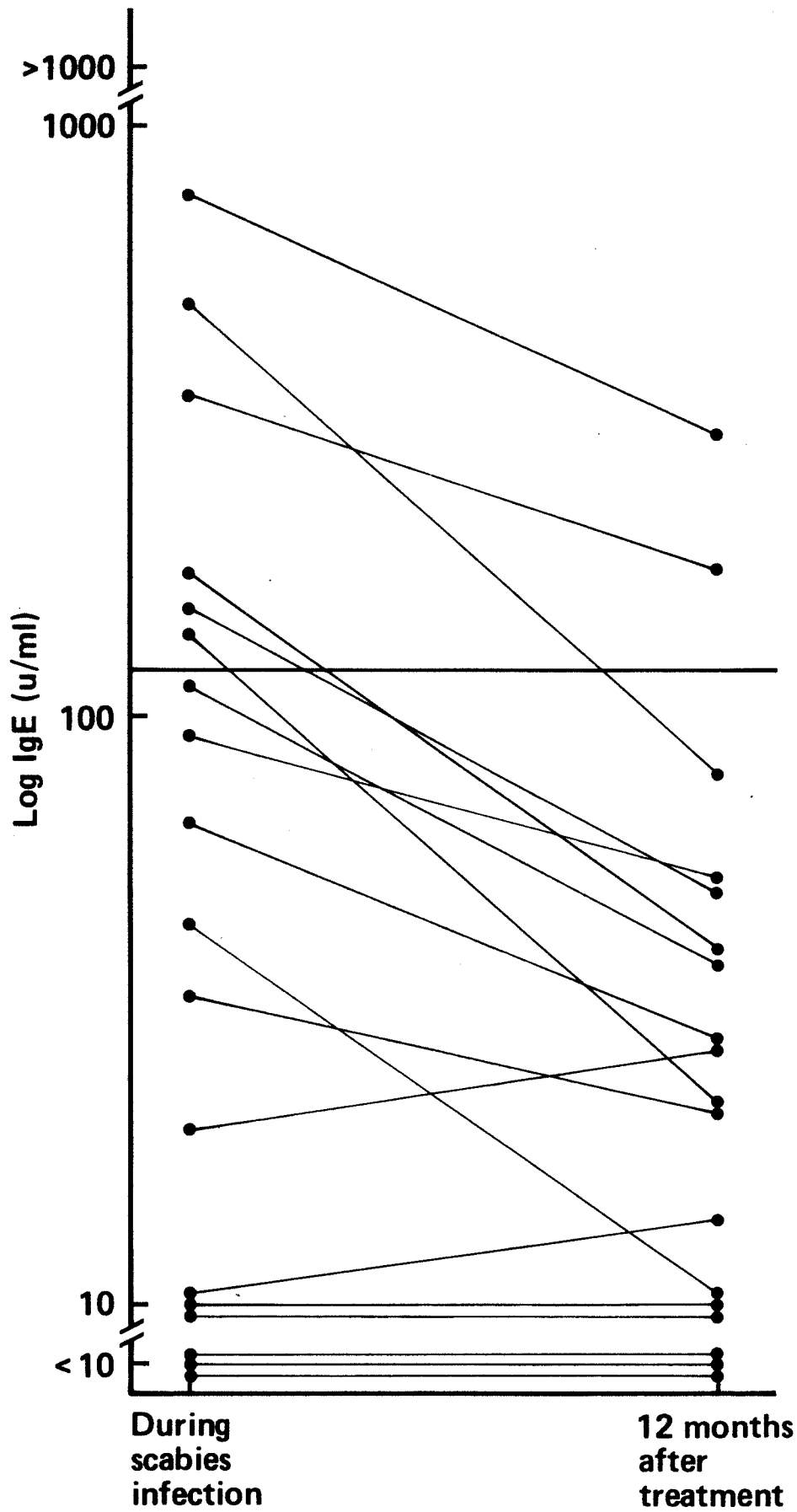


Fig. 1 C. IgE concentrations in 22 atopic patients with a positive RAST to *D. pteronyssinus* during scabies infection and 12 months after successful treatment.

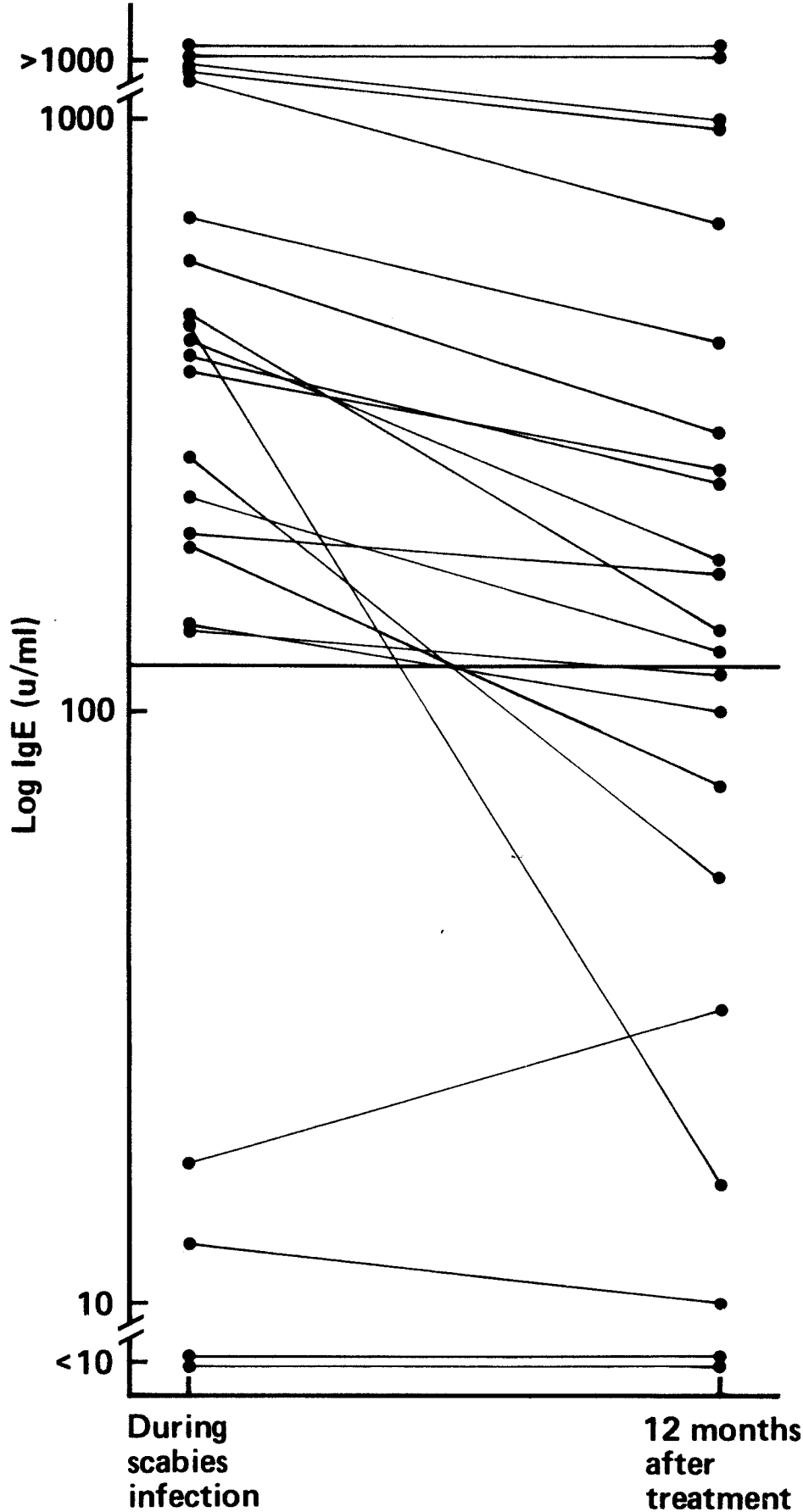


Fig. 1 D. IgE concentrations in 18 atopic patients with a negative RAST to *D. pteronyssinus* during scabies infection and 12 months after successful treatment.

tis, allergic rhinitis or extrinsic asthma. Latent atopic individuals, however, were also included. According to our criteria for latent atopy, in individuals with no personal history of atopy, the same specific antibody must be found both circulating and in the skin.

To avoid seasonal IgE variations, re-examination took place 12 months (Fig. 1 B, 1 C, and 1 D), and in some cases (Fig. 1 A), also 6 weeks and 21–24 months after treatment. The primary IgE measurements (2), however, were carried out at various times of the year. In order to eliminate atopic individuals, simultaneously, specific IgE antibodies were determined besides DP, also to house dust, horse dander, dog dander, cat dander, cow dander, *Cladosporium herbarum*, birch pollen, mugwort pollen, timothy pollen, cow's milk, cod, and egg white. These examinations, however, were not repeated.

Sera

Sera from all individuals were collected without additive prior to and 12 months after (in some cases 6 weeks and 21–24 months after) scabies treatment. They were stored at -20°C and used within 2 weeks after sampling.

Serum IgE

The IgE concentrations in serum were determined using the Phadebas-PRIST-test (Pharmacia AB, Uppsala, Sweden). The upper limit of normal IgE is 122 u/ml in adults (Nyco, Oslo, Norway).

Radioallergosorbent test (RAST)

The Phadebas RAST test (Pharmacia, Uppsala, Sweden) was used to quantify circulating IgE antibodies to DP. The RAST results were expressed in Phadebas RAST Classes 0, 1, 2, 3 and 4, according to recommendations from the manufacturer and employing a reference system established by running four reference sera (A, B, C and D) with reference discs during each assay in parallel with the unknown samples. In the present study Class 1 or more was considered to be a positive RAST result, although it is

possible that some Class 1 results represent non-specific reactions due to a raised IgE level.

Statistical methods

Statistical significance was calculated using three methods: sign test, Wilcoxon rank order test and Student's t-test.

RESULTS

For all 80 non-atopic patients the total serum IgE concentrations were lower 12 months after treatment than during scabies infection (Fig. 1 A and 1 B) ($P < 0.001$). This tendency could be demonstrated already after 6 weeks (Fig. 1 A). In most (29/40) of the atopic patients reduced IgE concentrations were evident 12 months later (Fig. 1 C and 1 D) ($P < 0.01$).

Likewise, the specific circulating IgE antibodies (RAST classes) to DP were reduced after treatment. These antibodies, like the IgE concentrations, were more markedly lowered in non-atopic patients, and all except one of the non-atopic patients had negative RAST to DP after 12 months (Table 1). The atopic patients, on the other hand, had only slightly reduced antibody-levels to DP after treatment (10/22) (Table 1).

DISCUSSION

Recently, we have demonstrated specific immunological hypersensitivity to scabies mite and that scabies infection stimulates the production of IgE antibodies (2, 3). The distinct decrease in total serum IgE concentrations and of specific circulating IgE antibodies to DP in all non-atopic patients after treatment, as compared with the values during scabies infection, supports the previous observations. The slight and inconsistent decrease of the same parameters in the atopic group may be due to a proportionately lower share of circulating IgE antibodies to the scabies mite. In a study of this phenomenon the most relevant approach would have been to quantify IgE antibodies to scabies as such. However, material for scabies RAST is not available at present.

TABLE 1. IgE ANTIBODIES TO DERMATOPHAGOIDES PTERONYSSINUS IN 18 NON-ATOPIC AND 22 ATOPIC PATIENTS WITH SCABIES, BOTH DURING INFECTION AND 12 MONTHS AFTER SUCCESSFUL TREATMENT

		RAST CLASS				
		4	3	2	1	0
NON-ATOPIC PATIENTS	DURING SCABIES INFECTION			9	9	
	AFTER TREATMENT				1	17
ATOPIC PATIENTS	DURING SCABIES INFECTION	5		14	3	
	AFTER TREATMENT	4	1	7	8	2

The immunological effect of scabies treatment parallels that of helminthic infections. Intestinal or systemic parasitism caused by helminths are important factors in stimulating the IgE production (7). In patients with capillariasis, total serum IgE concentrations decreased in the course of 18 months after treatment from six times the mean of the controls to near control levels in over 90% of patients (10), and similar observations were made in a patient with trichinosis, where IgE had returned to normal by the end of 6 months (11).

Studies in animals suggest that IgE antibodies may serve a protective function in parasitic diseases (12), and high IgE antibody activity has been associated with reduced worm populations (9). The development of hypersensitivity and partial immunity also to the scabies mite is supported by the experiments of Mellanby (8), showing that it is difficult to induce reinfection with scabies in persons who had had scabies some months previously and who had developed a high degree of hypersensitivity. In those cases the skin reacted so strongly and quickly (within 48 h) that most of the mites were destroyed. These observations could indicate that antibodies (circulating or bound in the skin) occur in scabies and that these antibodies may act as a protection from further infections with the scabies mite.

Our findings of a decrease in both total serum IgE concentrations and specific circulating IgE antibodies to DP 12 months after treatment, as compared with the values during scabies infection, suggest a loss of immunity during at least a period of 12 months after scabies infection. In an unpublished study we observed that eight reinfected patients, with a history of scabies 3 to 5 years previously, reacted as if they had got scabies for the first time. The fluctuations in the incidence of scabies, with about 15 years' interval between the end of one and the beginning of the next epidemic, may be explained by reduced immunity in the population.

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