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Fcγ-Receptors in Skin and Serum from Patients with Psoriasis, Before and After Therapy

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IgG-Fc receptors (FcR) are present on most immune competent cells. We have examined FcR in skin lesions from 8 patients with stationary plaque psoriasis and 12 patients with highly active psoriasis using MoAbs against FcR and binding of soluble immune complexes. FcR in serum were measured in ELISA. The patients were treated with cyclosporin (n = 5), acitretin (n = 7) and Goeckerman regimen (n = 8). As controls served 8 skin biopsies and 22 sera from healthy individuals. Highly active psoriatic lesions showed strongest activity for FcRI, II and III and immune complex binding. The FcR+ mononuclear cells were located perivascularly and along the dermo-epidermal junction. The FcR activity decreased in correlation to the improvement following therapy. Epidermal Langerhans cells (LC) were positive for FcRII and immune complex binding. FcR activity on LC decreased during therapy. Keratinocytes expressed FcRI and III, irrespective of disease activity and therapy. FcR levels were lower in sera from psoriatics than in controls, median 0.15 vs. 0.27 ($p < 0.01$), and not correlated to disease activity. In 4 patients the FcR levels increased during therapy. The reduced levels of FcR in psoriatic sera might be due to consumption in the skin or anti-FcR autoantibodies. ***Key words: IgG-FcR; cyclosporin; acitretin; Goeckerman.***

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IgG-Fc receptors (FcR) are present on most immune competent cells, being involved in antibody dependent cell-mediated cytotoxicity, in the release of cytokines, and in phagocytosis (1). Soluble FcR is a mediator of immunomodulation (2). There are at least 3 types of FcR, characterized using monoclonal antibodies (MoAbs). FcRI (CD16) is a strong receptor. FcRII (CD32) and III (CD64) are weak receptors (1). Dermal histiocytic cells express strong FcR activity (3). Epidermal Langerhans cells (LC) express weak FcR activity (FcRII)(4, 5). Keratinoctes (KC) have weak FcR activity (4), but react with both anti-FcRI and anti-FcRIII MoAbs (5).

In psoriatic lesions there is increased FcR activity, particularly on histiocytes (6) and in stratum corneum, but also on KC (7).

The aims of the present study were to:

1) characterize FcR in situ in psoriatic lesions,
2) examine soluble FcR in serum from patients with psoriasis,
3) examine the effect of different treatment modalities on the FcR.

MATERIALS AND METHODS

Skin biopsies and serum samples from 8 patients with stationary plaque psoriasis and 12 patients with highly active psoriasis were studied. The patients were treated with cyclosporin (n = 5), acitretin (n = 7) and Goeckerman regimen (n = 8). As controls served 8 skin biopsies and 22 sera from healthy individuals.

Cryostat sections were stained with:

1) anti-FcR MoAbs: 32.2 (anti-FcRI), IV3 (anti-FcRII), Leu11b (anti-FcRIII) (5) and B1D6 (against a weak unclassified FcR) (8).
2) soluble immune complexes of horseradish peroxidase (HRP) and rabbit IgG antibodies to HRP to detect functional FcR activity (6). Sera were examined for soluble FcR in ELISA with the MoAb B1D6.

RESULTS

Dermis

The FcR+ mononuclear cells were mainly located in the perivascular infiltrates, usually strongest in the dermal papillae, and in some lesions particularly strong along the dermo-epidermal junction. Highly active psoriatic lesions showed strongest activity for immune complex binding as well as reactivity with FcRI, II and III MoAbs. The number of FcR+ cells decreased in correlation to the improvement following therapy.

Epidermis

LC-bound immune complexes and were positive for FcRII. KC

Table I. *Serum levels of FcR (O.D.) in patients with psoriasis, before and after treatment*

Type of psoriasis	No. of patients	FcR (O.D.)			
		Before treatment		After treatment	
		Median	Range	Median	Range
Stable	8	0.175	0.141–0.955	0.147	0.133–0.950
Highly active	13	0.146	0.119–0.392	0.212	0.121–0.669
All patients	21	0.154	0.119–0.955	0.166	0.121–0.950
Controls	22	0.267	0.110–2.319		

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Table II. *Effect of psoriasis therapy on serum levels of FcR (O.D.)*

	Patients treated with					
	Cyclosporin		Acitretin		Goeckerman	
	(n = 5) Mean	Range	(n = 7) Mean	Range	(n = 9) Mean	Range
Before therapy	0.254	0.127–0.456	0.199	0.129–0.392	0.300	0.119–0.955
After therapy	0.320	0.130–0.493	0.281	0.130–0.669	0.305	0.121–0.950

expressed FcRI and III, strongest in highly active lesions. The FcR activity on both LC and KC decreased during therapy.

Sera

The FcR levels detected by B1D6 were lower in sera from patients with psoriasis than in controls, median 0.154 vs. 0.267 ($p < 0.01$), and were lower in highly active than in stable psoriasis, median 0.175 vs. 0.146 (Table I). In patients with highly active psoriasis, the median FcR levels increased during therapy, but after remission were still lower than in controls (Table I). This FcR increase occurred in patients treated with cyclosporine and acitretin (Table II) for highly active psoriasis.

DISCUSSION

The results showed that in psoriatic skin lesions there is an influx of FcR+ cells as well as increased FcR activity on KC and LC. In addition, the FcR activity is considerably increased in stratum corneum of psoriatic skin. The strongest FcR activity was detected in lesional skin from highly active psoriasis. The majority of the FcR+ mononuclear cells are histiocytes (6), which are important in the induction and control of immune responses. The large proportion of FcR+ histiocytes in the clinically most active psoriatic lesions sustains the concept of a local immune reaction early in the disease process (6, 9).

The reduced levels of FcR in psoriatic sera might be due to consumption in the skin or to anti-FcR autoantibodies (10). In systemic lupus erythematosus too, a reduced level of serum FcR has been found (11). During therapy, the FcR levels increased, but did not reach the levels in normal controls indicating an immunological defect not corrected by effective anti-psoriatic therapy.

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