Increased Plasma Norepinephrine in Psoriasis

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Free plasma catecholamines were measured by means of a standardized HPLC method in 50 adult patients with psoriasis and in 18 healthy volunteers. The concentrations of circulating norephinephrine were significantly higher in the psoriasis group (p < 0.005); by contrast only slight differences were found in the epinephrine and dopamine concentrations. The possible mechanisms leading to these changes are discussed. Key words: Psoriasis; Norepinephrine; High-performance-liquid-chromatography.

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We have recently published two reports describing increased plasma norepinephrine concentrations in severe atopic eczema (1, 2). Similar results were noted by us in patients with acute allergic asthma, a finding already reported by others (3). Finally we came to the conclusion that a large part of the elevated neurotransmitter may be released from the sympathicus by a disturbed regulatory mechanism (2).

Enhanced sympathetic activity may lead to im-

mune changes (4) and worsening of the clinical picture. Physiological norepinephrine release and availability seem to be important for normal T cell (and possibly B cell) function (5). We therefore raised the question: might immunobiological changes in psoriasis (6, 7) be related to increased norepinephrine levels. To tackle this question, we investigated the in vivo state of plasma catecholamines in patients with psoriasis and in healthy controls by means of a sensitive high-performance-liquid-chromatography (HPLC) method.

MATERIAL AND METHODS

Fifty adult patients (age range 17–45 years) with clinically proved psoriasis vulgaris (8) with more than 5 years' disease history, as well as 18 healthy volunteers having no history or sign of a skin disorder (age range 16–40 years) agreed to participate in this study. All patients avoided oral and topical corticosteroids and/or phototherapy for at least 2 months.

Venous blood samples were taken on Na-EDTA in the supine position at 9 a.m. after 10 min of bed rest. The concentration of plasma catecholamines was determined by reverse-phase HPLC with electrochemical detection (9). Chromatographic separation was carried out on a C-18 plasma catecholamine column (5 \times 150 mm, spherical particle size: 5 μ m) after Al_2O_3 extraction. Equipment, standardized method and reagents were supplied by Waters,

Table I. Catecholamine levels in psoriasis patients and healthy controls

	Epinephrine (pg/ml)	Norepinephrine (pg/ml)	Dopamine (pg/ml)	
Psoriasis patients (n=50)	47±20	305±95	24±16	
Controls (n=18)	43±22	174±56	17±14	
Significance Student's t-test	NS	p<0.005	NS	

Millipore, FRG. Results are expressed in pg/ml and the standard error of two determinations was less than 15%.

RESULTS AND DISCUSSION

In our study, plasma norepinephrine concentrations were significantly elevated in psoriasis patients (p < 0.005; Table I). No clear-cut correlation between norepinephrine levels and extent or activity of the skin disease was apparent. Slightly increased levels of epinephrine and dopamine were recorded in the same subjects, but the difference was statistically insignificant when compared with the control group (Table I). This pattern parallels the increase in norepinephrine in atopic eczema patients (1) where neither elevated dopamine- β -hydroxylase (DBH) activity, nor decreased catechol-0-methyltransferase and phenylethanolamine-methyltransferase activities were found (1, 2).

Type B monoamine oxidase activity in platelets of psoriasis patients is significantly depressed (10). Types A and B monoamine oxidase have only small differences in their substrate specificity. Type A monoamine oxidase activity is therefore believed to be lowered too. All tested substrates of types A and B monoamine oxidase (serotonin, tyramine, dopamine, epinephrine, norepinephrine) in psoriatic plasma proved normal (11), except norepinephrine. We therefore conclude that the monoamine oxidases are not responsible for the abnormal norepinephrine concentrations in psoriatic patients.

However, we cannot rule out the possibility that the elevated transmitter may be released from and/or taken up by the sympathicus via a disturbed regulatory mechanism. An increased cellular Ca²-inflow might be responsible for the greater release of norepinephrine, in analogy to the Ca²+-induced histamine outflow from mast cells or basophils. Psychogenic factors, biogenic amines, food additives or

other environmental factors (12) are possible triggers of increased cellular Ca²⁺ inflow. In this context, a raised norepinephrine level may lead to immune changes (4, 5) and worsening of the clinical picture in psoriasis patients.

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