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Monoamine- and Diamine Oxidase Activities in Psoriasis

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Monoamine- and diamine oxidase activities were measured by a sensitive photometric assay in 25 psoriasis vulgaris patients. Results were compared with plasma histamine values determined fluorimetrically. Increased plasma histamine levels were associated with significantly lowered diamine—and type B monoamine oxidase activities in platelet-rich plasma of the psoriasis patients. Our data suggest that cofactor levels and/or inhibiting factors are responsible for the observed monoamine- and diamine oxidase activities. Key words: Histamine, MAO-B; DAO.

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Attempts to use the activity of the mitochondrial type B monoamine oxidase of the platelets to diagnose mental disease are common, but the results remain contradictory. All attempts to use diamine oxidase for the diagnosis of certain diseases have been unsuccessful until now, because of uncertain distinction between the normal and pathological range.

The preferred substrates of type B monoamine oxidase are benzylamine, 2-phenylethylamine, dopamine, tyramine and, with lower activity, tryptamine. *N*-methylhistamine is also oxidized by type B monoamine oxidase. Diamine oxidase is active on shortchain aliphatic diamines such as putrescine and cadaverine. Diamine oxidase is also a histamine catabolizing enzyme.

We have recently reported low mono- and diamine oxidase activities in atopic eczema patients (1). We now address the question whether these enzymes are also of significance in the pathogenesis of psoriasis and furthermore if plasma histamine levels are related to monoamine oxidase B (MAO-B) and diamine oxidase (DAO) activities.

MATERIAL AND METHODS

Twenty-five patients (age range 17–43 years) with clinically proved psoriasis vulgaris (2) of more than 5 years' duration, as well as 14 healthy volunteers having no history or sign of a skin disease (age range 16–39 years) agreed to participate. The patients avoided all steroid and/or phototherapy for at least 2 months.

Platelet-rich plasma (PRP) was obtained by centrifugation of stabilized (EDTA) blood at 53 g (600 rpm) for 10 min at 20°C. The oxidases were measured *ad modum* Köchli & Wartburg (3), with minor modifications. 0.6 ml peroxidase buffer (8.3 mg peroxidase in 100 ml of 0.1 M sodium phosphate, pH 7.15), 0.2 ml PRP and 10 µl 10% Triton X-100 were mixed; after 5 min, 0.2 ml 0.25 mM 2.7-dichlorofluorescein diacetate dissolved in 0.01 N NaOH and 10 µl 1 mM benzylamine (for MAO-B) or 10 µl 50 mM putrescine (for DAO) were added and mixed. The absorbance at 502 nm was recorded after 15–25 min in a Shimadzu UV-160 spectrophotometer at 20°C. Histamine was measured in EDTA plasma *ad modum* Shore (4), using a Perkin-Elmer LS-2 filter fluorimeter.

O-phtalaldehyde, histamine, benzylamine, putrescine, Triton X-100 were obtained from Sigma, München, FRG; 2.7-dichlorofluorescein diacetate from Serva Heidelberg, FRG and horseradish peroxidase from Boehringer, Mannheim, FRG.

Table I. Monoamine and diamine oxidase activities in platelet-rich plasma of psoriasis patients and healthy controls

1,11.2-12) #E	Type B monoamine oxidase (mmol min ⁻¹ 1 ⁻¹)	Diamine oxidase (mmol min ⁻¹ 1 ⁻¹)	Histamine (μg 1 ⁻¹)
Psoriasis vulgaris	0.242 ± 0.095 ($n=25$)	0.231 ± 0.101 $(n=24)$	5.41 ± 2.05 ($n=25$)
Controls	0.394 ± 0.091 ($n = 14$)	0.535 ± 0.133 ($n=13$)	2.25 ± 1.00 $(n=13)$
Significance Student <i>t</i> -test	p<0.05	p<0.001	p<0.001

RESULTS AND DISCUSSION

The investigations show reduced MAO-B and DAO activities in platelet-rich plasma of psoriasis patients, when compared with control subjects (Table I). The difference was highly significant for the first step histamine catabolizing enzyme, DAO (p<0.001) and significant for the methylhistamine catabolizing enzyme MAO-B (p<0.05). Concomitantly with reduction of MAO-B and DAO activities, plasma histamine levels increased (Table I). Our results suggest that low MAO-B and DAO activities may account for increased histamine levels of endogeneous or exogeneous (5) origin in psoriatic patients.

The cofactor concentrations of MAO-B (6) were reduced but for DAO (7) were almost normal (manuscript in preparation). We therefore conclude that, contrary to MAO-B activity, some other reason such as the cofactor level must be responsible for the lowered activity of DAO. Biogenic amines, food additives, or drugs (5, 8) are the most probable candidates for inhibition of the enzyme. Investigations concern-

ing DAO inhibitors in psoriasis are in progress in our laboratory.

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Dietary Supplementation with a Combination of n-3 and n-6 Fatty Acids (Super Gamma-Oil Marine) Improves Psoriasis

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Psoriasis may improve during dietary supplementation with fish oil containing n-3 fatty acids including eicosapentaenoic acid. In the present study 17 psoriatic patients were treated with Super Gamma-Oil Marine containing a combination of n-3 and n-6 fatty acids (linoleic acid and gammalinolenic acid). After 4 months, excellent improvement was observed in 2 patients, moderate improvement in 8, mild improvement

in 4, and no improvement in 3 patients. These results may indicate that a combination of n-3 and n-6 fatty acids is useful for the treatment of psoriasis. However, controlled studies including more patients are warranted

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