

Cofactor levels of mono- and diamine oxidase in atopic eczema

G. IONESCU & R. KIEHL

Research Department, Spezialklinik Neukirchen, W. Germany

We have recently shown that increased plasma histamine levels were associated with significantly lowered diamine and type B monoamine oxidase activities in platelet rich plasma of atopic eczema patients. We now report almost normal cofactor (pyridoxal phosphate and Cu^{2+}) levels for diamine oxidase but lowered cofactor (flavin adenine dinucleotide and Fe^{2+}) levels for type B monoamine oxidase.

Key words: atopic eczema; copper; diamine oxidase; flavin adenine dinucleotide; histamine; iron; monoamine oxidase; pyridoxal phosphate.

Accepted for publication 4 February 1989

We have recently reported (2) that increased plasma histamine levels were associated with significantly lowered diamine and type B monoamine oxidase activities in platelet rich plasma of atopic eczema (AE) patients. There may be two obvious reasons for the lowered enzyme activities, supposing the enzymes themselves to have no defect: cofactor deficiency or presence of inhibiting factors.

The purpose of this study was to evaluate the cofactor levels of the histamine catabolizing enzymes, monoamine and diamine oxidase in AE patients and to compare them with the cofactor levels of control subjects.

MATERIAL AND METHODS

Twenty patients with clinically proved atopic eczema (1) (age 15-44 years) and 11 healthy volunteers with no allergic history (age 16-39 years) consented to participate. All patients avoided any steroid or antihistaminic treatment for at least 3 months before admission.

Type B-monoamine and diamine oxidase activities in platelet rich plasma as well as

plasma histamine were estimated as previously described (2). Serum copper (11), serum pyridoxal phosphate (8), serum iron (5), EDTA-blood haemoglobin (9) and flavin adenine dinucleotide (FAD) in EDTA-blood (6) were measured by routine procedures as outlined.

RESULTS AND DISCUSSION

The cofactor levels for monoamine oxidase, flavin adenine dinucleotide (FAD) and iron (10), were lowered in AE-patients when compared with control subjects (Table 1). Iron values were at the bottom of the normal range. Concomitantly with lowered iron values haemoglobin values were almost normal (Table 1), suggesting that AE patients have a small iron storage deficiency. FAD in these patients was significantly lowered ($P < 0.005$) when compared with a control group (Table 1). Monoamine oxidase activity may be restored by iron and vitamin B₂ supplementation.

The cofactor levels for diamine oxidase, pyridoxal phosphate and copper (7), were al-

TABLE 1

Monoamine oxidase activity, cofactor level and haemoglobin concentration in atopic eczema

	Type B monoamine oxidase (mmol min ⁻¹ l ⁻¹)	FAD (µg/l)	Iron (µg/dl)	Haemoglobin (g/dl)
Atopic eczema patients (n)	0.223±0.110 (19)	79.6±13.1 (16)	m 94.6±25.8 (7) w 73.6±27.0 (13)	m 13.6±1.5 (7) w 13.2±0.9 (13)
Controls (n)	0.371±0.085 (11)	132±42 (11)	m 130±50* w 110±50	m 15.5±2.5* w 14±2
Significance, Student t test	P<0.05	P<0.005	NS	NS

*generally accepted normal values

TABLE 2

Diamine oxidase activity and cofactor level in atopic eczema patients and healthy controls

	Diamine oxidase (mmol min ⁻¹ l ⁻¹)	Copper (µg/dl)	Pyridoxal phosphate (µg/l)
Atopic eczema patients (n)	0.270±0.089 (18)	125.3±28.9 (18)	12.1±9.4 (17)
Controls (n)	0.511±0.125 (10)	115±50*	10.8±7.2 (11)
Significance, Student t test	P<0.001	NS	NS

*generally accepted normal values

most normal in the atopic group (Table 2). We therefore concluded that, contrary to monoamine oxidase activity, a reason other than the cofactor level must be responsible for the lowered activity of diamine oxidase. Biogenic amines, food additives, or drugs (3, 4) are the most probable candidates for inhibition of the activity. Investigations concerning diamine oxidase inhibiting factors are in progress in our laboratory.

ACKNOWLEDGEMENT

We thank laboratory Dr. Hofmeister, 8480 Weiden/Opf., West Germany for providing us with the HPLC analyses of FAD and pyridoxal phosphate.

REFERENCES

1. Hanifin, J. M. & Lobitz, W. C.: Newer concepts of atopic dermatitis. *Arch. Dermatol.* 113, 663-670, 1977.

2. Ionescu, G. & Kiehl, R.: Monoamine- and diamine oxidase activities in atopic eczema. *Allergy* 43, 318-319, 1988.

3. Paul, E.: Allergische und pseudoallergische Hautreaktionen auf Nahrungsmittel und Nahrungsmittelbestandteile. *Med. Welt* 38, 847-851, 1987.

4. Sattler, J. & Lorentz, W.: Nahrungsmittel induzierte Histaminose. *Münch. Med. Wochenschr.* 129, 551-557, 1987.

5. Siedel, J., Wahlefeld, A. W. & Ziegenhorn, J.: Improved, Ferrozine®-based reagent for the determination of serum iron (transferrin iron) without deproteinization. *Clin. Chem.* 30, 975, 1984.

6. Speek, A. J., van Schalk, F., Schrijver, J. & Scheurs, W. H. P.: Determination of the B₂ vitamin flavin - adenine dinucleotide in whole blood by high performance liquid chromatography with fluorometric detection. *J. Chromatogr.* 228, 311-316, 1982.

7. Summary of an international workshop on diamine oxidase held at the department of biogenic amines. Polish Academy of Sciences, Lodz, Poland, June 7-8, 1979, *Agents Actions* 11, 3-8, 1981.

8. Ubbink, J. B., Serfontein, W. J. & Villiers, L. S.: Stability of pyridoxal-5-phosphate semicarbazone: applications in plasma vitamin B₆ analysis and pop-

- ulation surveys of vitamin B₆ nutritional status, J. Chromatogr. 342, 277-284, 1985.
9. Van Kampen, E. J. & Zijlstra, W. G.: Standardization of haemoglobinometry. II. The haemoglobincyanide method, Clin. Chem. Acta 6, 538, 1961.
10. Youdim, M. B. H. & Holzbauer, M.: Physiological and pathological changes in tissue monoamine oxidase activity, J. Neural Transm. 38, 193-229, 1976.
11. Zak, B.: Simple procedure for the single sample determination of serum copper and iron. Clin. Chim. Acta 3, 328-334, 1958.

Address:

Dr. G. Ionescu

Spezialklinik Neukirchen

8497 Neukirchen

West Germany

This document is a scanned copy of a printed document. No warranty is given about the accuracy of the copy. Users should refer to the original published version of the material.