

Letter to the Editor

To the Editor,
Abnormal plasma catecholamine levels in severe atopic eczema (AE) were recently reported by us in a previous issue of your journal (1). In our study plasma norepinephrine was significantly elevated in severe AE patients $P < 0.005$. Slightly decreased levels of epinephrine and slightly increased levels of dopamine were recorded in the same subjects, but the difference was statistically insignificant when compared with the control group.

Norepinephrine elevation due to high disease stress could be excluded as concomitantly raised epinephrine levels were not found. We also gave up the assumption of an elevated dopamine-B-hydroxylase (DBH) activity because we found almost normal DBH activity in the atopic group (1).

Type B monoamine oxidase activity in platelets of AE patients is significantly lowered (2). Two of their preferred substrates are dopamine and tyramine. The cofactor levels for type B monoamine oxidase, flavin adenine dinucleotide (FAD) and iron, were lowered in AE patients compared with control subjects (3). Type A and B monoamine oxidase have only small differences in their substrate specificity. Some of the preferred substrates of type A monoamine oxidase are dopamine, norepinephrine, epinephrine, tyramine and serotonin. Type A monoamine oxidase activity is therefore supposed to be lowered too. All substrates of type A and B monoamine oxidase (serotonin, tyramine, dopamine, epinephrine, norepinephrine) measured were normal, except norepinephrine. We conclude therefore, that the monoamine oxidases are not responsible for abnormal norepinephrine concentrations in AE patients.

We suggested decreased catechol-O-methyltransferase- and phenylethanolamine-methyltransferase activities as responsible for elevated norepinephrine concentration (1). We have now measured erythrocyte catechol-O-methyltransfe-

rase activity of AE patients and healthy controls using an isotopic assay (4). Slightly decreased levels of catechol-O-methyltransferase were recorded in AE patients, but the difference was statistically insignificant compared with the control group (Table 1). Moreover, with raised norepinephrine concentrations, a compensatory increase of catechol-O-methyltransferase activity was apparent. Phenylethanol-amine-methyltransferase activity should be normal since epinephrine level is normal.

Table 1
Norepinephrine level and catechol-O-methyltransferase (COMT) activity in atopic eczema patients and healthy controls

	Norepinephrine (pg/ml)	COMT (dpm- blank/0.5)*
Atopic eczema patients (age: 18-48; n = 17)	383±144	3275± 930
Controls (age: 19-40; n = 18)	174± 56	3934±1352
Significance		
Student t test	$P < 0.005$	NS

*blank = 200 dpm, 0.5 = 50 % extraction of 4-hydroxy-3-methoxybenzoic acid into the organic phase.

Summarizing, the only remaining possibility is that the elevated transmitter may be released from and/or taken up by the sympatheticus in a disturbed regulatory mechanism. An increased cellular Ca^{2+} -inflow could be responsible for the enhanced release of norepinephrine, in analogy to the Ca^{2+} -induced histamine outflow from mast cells or basophils. A defect membrane transport protein may account for lowered mediator uptake. Research on this topic is in progress in our laboratory.

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