PLATELET SEROTONIN AND PLATELET MAO ACTIVITY IN ALZHEIMER'S DISEASE

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SUBJECTS & METHODS:
The diagnosis of the probable Alzheimer’s disease according to NINCDS-ADRDA criteria was established by two psychiatrists according to ICD-10 and DSM-IV-TR criteria. A total of 34 AD patients (14 men; 20 women) and 75 healthy controls (36 men; 39 women) were analyzed.

Control group consisted of 36 male and 39 female drug-free healthy subjects (mainly medical stuff), with no history of psychiatric illness.

All subjects had a medical evaluation and routine laboratory screening tests prior to entry into the study, gave written informed consent to participate in the trial.

At baseline (before treatment, i.e. after 1 week of washout), a forearm vein was cannulated for blood sampling at 08.00 a.m., after an overnight fasting. Blood samples (8 ml) were drawn in a plastic syringe with 2 ml of acid citrate dextrose anticoagulant. Platelet rich plasma (PRP) was obtained after centrifugation of PRP. Platelet 5-HT concentration was determined by the spectrofluorimetric method, as previously described (Mück-Šeler et al., 1996a) using Aminco-Bowman spectrofluorimeter on an exciting wave length of 345 nm and emitted wave length of 380 nm. Platelet MAO activity was determined spectrofluorimetrically using kynuramine as a substrate, by a slight modification of the method of Krajl (1965), as previously described (Mück-Šeler et al., 2002). The measurement of 4-hydroxyquinoline (4-HOQ) fluorescence, a product of kynuramine, was performed on Aminco-Bowman spectrofluorimeter, with an exciting wavelength of 310 nm and emitted wavelength of 380 nm. Platelet protein levels were measured by the method of Lowry et al. (1951).

All data (expressed as mean ± S.D.) were evaluated by one-way analysis of variance (ANOVA), followed by Tukey’s multiple comparison test.

RESULTS

CONCLUSION:
Our preliminary data, from this ongoing study, support the thesis that platelet serotonergic markers (5-HT and MAO) are altered in AD patients, and may play a role in the etiopathogenesis of this disease.

REFERENCES:

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