We have examined SER functioning (a) by measuring basal and stimulated concentrations of intraplatelet free calcium ([Ca2+]i), in response to thrombin (1 unit/ml), using fura-2 and (b) by measuring the specific activities of calcium dependent enzymes such as, Ca2+/Mg2+-ATPase, Mg2+-ATPase and Ca2+-ATPase, using methods previously reported for determination of inorganic phosphate. Our results (Table 1) show that, there was a significant increase in the percentage of [Ca2+]i, released above the basal levels with thrombin stimulation in AD platelets compared to controls. This was partly due to a lower basal level of [Ca2+]i in AD platelets compared to controls. We have also found, that the specific activities of Ca2+-ATPase, Mg2+-ATPase and Ca2+/Mg2+-ATPase is significantly reduced in AD platelets compared to controls (Table 1).

These data suggest that calcium homeostasis is altered in AD platelets. This is comparable to the findings in AD fibroblasts.3 We might be important in the CNS pathophysiology of this disorder.

Table 1. 
<table>
<thead>
<tr>
<th>Basal Stimulated Increase</th>
<th>Mg2+</th>
<th>Ca2+/Mg2+-</th>
<th>Ca2+-</th>
<th>ATPase (mmol/mg protein)</th>
</tr>
</thead>
<tbody>
<tr>
<td>ALS n=19</td>
<td>n=19</td>
<td>n=24</td>
<td>n=24</td>
<td></td>
</tr>
<tr>
<td>149822</td>
<td>548550</td>
<td>306195**</td>
<td>7.460 4</td>
<td>9.640±6* 2.210±3*</td>
</tr>
<tr>
<td>COM n=10</td>
<td>n=10</td>
<td>n=16</td>
<td></td>
<td></td>
</tr>
<tr>
<td>108423</td>
<td>476470</td>
<td>13448</td>
<td>8.816 2</td>
<td>13.818± 4.590±9</td>
</tr>
</tbody>
</table>

Values are the Means±SEM.

*p<0.05, **p<10^{-5}; Significantly different from controls

References

**DIFFERENT PATTERNS OF CSF MONOAMINE METABOLITES IN OLD-AGE DEMENTIAS.**

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Many investigations have been carried out in recent years in an attempt to clarify the etiopathogenetic role of neurotransmitter deficiencies in dementia disorders. With regard to "ex vivo" studies on bio- 
genome amines, which have importance in regulating cognitive and motor functions, attention has been focused on CSF levels of the key breakdown products of these neurotransmitters: homovanillic acid -HVA- and 3-methoxy-4-hydroxyphenyl -ethyl- 
glycol (MHPG) for dopamine, homovanillic acid (HVA) and 5-hydroxy-tryptophan (5-HT) for serotonin and 3-methoxy-4-hydroxyphenyl-ethyl- 
glycol (MHPG) for norepinephrine, since they reflect the activity of these neurotransmitters. With the aim of better understanding the pathogenetic usefulness of these biological markers in dementia disorders, we have thus measured CSF levels of the main metabolites (HVA, MHPG, 5-HT, dopamine, norepinephrine) in patients with early-onset Alzheimer's disease (e-AD, n. 12), late-onset AD (1-AD, n. 13), vascular dementia (VD, n. 13), single senile dementia (SSD, n. 10) and twelve elderly controls. Psychobehavioural assessment was also carried out by means of MMSE and GBS Rating Scale for Dementia. Mean MMSE levels did not differ from controls among the groups considered, while 5- 
HIAA was slightly lower (0.10>p>0.05) in e-AD, 1-AD and VD; HVA was consistently decreased (p<0.02) in e-AD and 1-AD, showing a slight reduction (0.10>p>0.05) in VD. No variation was found in SSD.

Negative and differently distributed correlations were observed between psychochemical and psychobehavioural parameters and performance in subjects who did not show any relationship between these variables was documented in e-AD. These results confirm that CSF levels of monoamine metabolites are of scarcer diagnostic value but reinforce the evidence of the clinical heterogeneity of old-age dementia and the potential importance of these clinical-biological studies in view of differentiated therapeutic treatments.

**CORRELATIONS BETWEEN AND AMONG PURINES AND PURINE METABOLITES IN ALZHEIMER'S DISEASE.**

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Through the utilization of large, multiparameter data bases containing sufficiently reliable information, data which may seem chaotic can be used in the definition of normal metabolism and the altered state metabolism associated with specific disorders. Studies of patients with neuropsychiatric disorders suggest that dietary levels of tryptophan and tyrosine profoundly affect the levels of various neurotransmitters. Deficiencies in purine metabolism have been linked to neurochemical lesion in Parkinson's Disease, while variations in purine metabolites have been implicated in Alzheimer type dementias.4

A multiparameter data base has been created utilizing a Coulonchem® Electrode Array System. Approximately 1000 Alzheimer, Huntington, Parkinson, control tissue and CSF samples comprise this data base. We have analyzed these data for potential metabolic interactions between the purine, tryptophan and tyrosine pathways. A strong correlation has been observed between levels of tyrosine and xanthine.

Tyrosine/xanthine ratios were found to be significantly decreased in the putamen and A4 of the Parkinson samples. Guaanosine levels in putamen, A20, A21, A9, and A4 in Alzheimer samples were decreased approximately 50 percent of control values. Alterations in the metabolites in the few reports that have been associated with changes in the tyrosine/xanthine pathways. These data suggest a relationship between purine and aromatic amino acid metabolism in certain central disease disorders.