In both the control and Alzheimer's groups, significant inhibitions of adenylly cyclase activity were produced by somatostatin (2P = 0-012, Wilcoxon's matched-pairs signed ranks test) and NPY (2P = 0-012). In the Alzheimer's group, however, the degree of somatostatin enzyme inhibition was significantly lower than that for the control group. This difference was seen when both the absolute decreases in cAMP production and the percentage decreases in basal activity were compared (table). Furthermore, there was a larger spread in the observed somatostatin inhibitions for the Alzheimer's group (compare the SEM values to the mean values in the table). In three of the Alzheimer's cases, essentially no somatostatin inhibition of adenylly cyclase activity was found, whereas the highest percentage inhibition found in the control cases was 12%. For the control group, there were found to be no correlations (Spearman's rank) between either basal activity, somatostatin or NPY inhibitions of basal activity and age or postmortem time.

The levels of 123I-Tyr1-somatostatin-14 binding in the control and Alzheimer's disease groups were not significantly different (table), a finding in accordance with some but not all investigators (1991;1:45). These data suggest that the lower degree of somatostatin inhibition of adenylly cyclase activity in the Alzheimer's group was not due simply to a lower receptor density. Important to note, was that the degree of NPY enzyme inhibition was similar in both groups, indicating that the observed deficit was specific to the somatostatin system and was unlikely to be a result of such factors as agonal status or drug treatment of the disease cases.

This study showing a reduced somatostatin modulation of adenylly cyclase activity in Alzheimer's disease is the first to our knowledge demonstrating a functional deficit of somatostatin receptor integrity in this disorder. Further experiments will be necessary to determine the mechanism underlying this dysfunction, such as for example studying the integrity of somatostatin receptor—

**G**-protein—adenylly cyclase interactions. It will also be interesting to determine whether this dysfunction is found in other brain regions and different degenerative diseases. Alzheimer's disease pathology and whether it is important for the cognitive decline seen in the disorder.

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**Letters to the Editor**


Although plasma DBH activity is a peripheral blood index, it more directly reflects the metabolism of catecholamines than other indices such as the metabolite MHPG levels. Determinations are needed to evaluate this and indirect approaches of central noradrenergic activity. Plasma DBH activity may reflect the state of releasable stores of the enzyme in sympathetic nerve endings.

It may be concluded that plasma DBH activity is decreased in depressed patients when compared with age-matched controls. Whether this decrease is observed in every patient or in a subgroup remains a prospective for future studies.

The most interesting and original result is the increase in plasma DBH activity in euthymic patients, that is, after five weeks of treatment with tricyclic antidepressants. The respective role of antidepressant drugs and thymic improvement remains unclear. However, the level of plasma DBH activity remains significantly (p<0.05) lower in euthymic patients than controls.

DBH activity is a genetic component in the pathophysiology of depressive states could be suggested since altered plasma levels of DBH may reflect a genetic susceptibility. Finally, further studies on DBH activity in plasma may provide an interesting for the role of DBH in the development and pathogenesis of depression.

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