Form 2-4-2

Japan-US Brain Research Cooperation Program
Group Joint Study Report
[field: Research on neuromechanism of emotion and memory]

1. The Representative of Group Joint Study:
Nobumasa Kato: Professor, Department of Neuropsychiatry, Graduate School of Medicine, University of Tokyo

2. Project Title:
Influence of stress on the neural plasticity in hippocampus

3. Japanese Investigator's Name, Title, Affiliation and Phone Number:
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4. U.S. Investigator's Name, Title, and Affiliation:
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5. The Term of Research: From April 4, 2002 to March 31, 2003 (3 years)

6. Abstract, Result and Significance of Research (300 Words):

We have investigated the stress-induced hippocampal changes including intracellular signaling pathways and neural immune systems, and have reported that these changes might be involved in the hippocampal neuroplasticity. To advance our understanding of the influence of stress on these hippocampal changes, we have discussed with several research groups in U.S. as noted above. Dr. Sadamatsu and Dr. Tsunashima have collaborated with Prof. McEwen, who is the chief of U.S. side, and have investigated the changes in hippocampal neurogenesis and neuronal death induced by trimethyltin (TMT). After TMT administration, neurogenesis was significantly decreased in the dentate gyrus at 5-7 d after treatment, and subsequently returned to basal level at 14-28 d. In comparison, considerable neuronal loss in the hippocampus was observed at 3 d after treatment, and lasted more than 3 weeks. They also revealed that these changes were involved in a transient elevation of circulating corticosterone. Dr. Sadamatsu presented these results at the 32nd meeting of Society for Neuroscience held in Orlando.

Dr. Kohda has examined the stress-induced synaptic plasticity in the hippocampus with Dr. Sawa, a collaborator of U.S. side. They studied the time course of effects of the behavioral stress on long-term synaptic plasticity in the hippocampal CA1 region, and revealed that the behavioral stress should have acute and late-onset effects on long-term synaptic plasticity in CA1, which might result from the higher corticosterone level caused by the stress exposure. Dr. Kohda also presented their results in Orlando.

Further, we held several stress-related symposiums in Tokyo during last summer, and published our results as a feature of several scientific journals.

7. The Others (Practical Issues, Special Mention Matters):