INTERACTION OF AN EXTRACELLULAR CALCIUM-BINDING PROTEIN WITH SYNAPTOSOMAL ORGANELLES AFTER VESTIBULOMOTOR TRAINING AND CLASSICAL AVOIDANCE CONDITIONING IN GOLDFISH. Rupert Schmidt University of Frankfurt, F.R.G.

Ependymins are dimeric glycoproteins sharing several properties, such as low pI and a high Phe:Tyr ratio, with other calcium-binding proteins. By radioimmunoassay (RIA) and immunofluorescence they were localized to pyramidal neurons in the goldfish optic tectum and rat hippocampus. Following subcellular fractionation, large amounts of ependymins were recovered from the cytoplasmic compartment and a fraction collected from the extracellular fluid of goldfish brain (ECF). The synaptosomal fraction contained small amounts of ependymins, mainly ependymin γ . γ is derived from the β-subunit of ependymin molecules by the hydrolytic action of an extracellular metallo-protease. Subcellular organelles recovered in the synaptosomal fraction incorporate intraventricularly injected 125-I-labeled ependymin γ . RIA measurements revealed that ependymin concentrations in the ECF decreased temporarily in fish that had learned a vestibulomotor task, before they increased above control levels. Ependymin concentrations in the cytoplasm increased immediately after learning. Intraventricularly injected anti-ependymin antisera not only prevent recall of this task, but also of classical avoidance conditioning in the shuttle-box, when injected between 0.2 and 24 h after acquisition. Goldfish fleeing spontaneously the light stimulus, which was used for conditioning, were not affected (active control).

It is suggested that a decrease in extracellular calcium in the synaptic cleft after learning might induce a conformational change or prevent proteolysis of secreted ependymin molecules, thereby triggering their interaction with synaptic membranes. (Supported by the Deutsche Forschungsgemeinschaft, Schm 478)