The role of interactions of platelets with brain-specific neuronal glycolipids in the modulation of neuronal functions during neurological disorders

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In the intact CNS platelets are separated from the neurons and glial cells by the blood-brain barrier (BBB) structure. However, during pathological condition such as the trauma, infection, neurodegeneration, inflammation BBB become compromised leading to direct interaction of neurons with platelets. We have previously found that brain-specific sialated gangliosides within neuronal lipid rafts directly activated platelets.

Currently we investigate the possible role of platelet-lipid raft interactions in the modulation of neuronal activity in mouse model of epilepsy.

We found that the seizure durations were significantly reduced in wild-type (WT) mice with depleted platelets. We considered serotonin release a possible link between platelets and neural activity since blood platelets are a major source of this neurotransmitter in a periphery. The overall seizure intensity differs was lower in mice with inhibited 5-HT in platelets in comparison to control. Platelet intracranial injection causes seizures comparable with PTZ-induced seizures.

The activity of neurons was increased when neuronal cells were co-incubated with platelets, both in a primarily neural culture and in brain slice organotypic cultures. During the interaction with neuronal lipid rafts platelet do indeed release serotonin which was confirmed by LC/MS experiments.

Thus, our data indicate the possible role of platelet-lipid raft interactions in a modulation of neural activity. The possible agent of interaction between platelets and neurons is serotonin (Pic. 1). We propose the platelet-neural interaction to be a potential target in treating epilepsy and other neurological disorders.