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Schizophrenic dysintentionality based on a severe disorder of glial-neuronal interaction

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The pathophysiology of schizophrenia may essentially be caused by a loss of the glial boundary setting f tripartite synapses (Mitterauer, 2003; 2005). Astrocytes are producing non-functional proteins or transm the synaptic information flux is uninterrupted leading to a generalization of information processing in ne Mutations in astrocytes may be responsible for this disorder. Due to the generalization of information pr neuronal networks become compartmentless, incapable to compute environmental information with rega ontological domains (specific objects, individuals, etc.). Therefore, schizophrenic cognition always deals case (e.g. not with an individual person, but with mankind). One can also say that patients with schizopl suffering from a loss of self-boundaries. The typical symptoms are delusions, hallucinations, thought dis and affective flattening.

However, what may in parallel occur in the panglial syncytium? In further elaborating my theory of glial- interaction, I have hypothesized that the intentional or action programs of the brain my be generated in syncytium (Mitterauer, 2006). Based on a formal model it can be shown how glial syncytia compute in a combinatorial manner cycles of various lengths via gap junctions. These cycles are transferred in triparti the neuronal system. The neuronal system tests these intentional programs with regard to their feasibili environment. In feeding back the feasibility of intentional programs to the glial syncytium, learning proc

For the clinically experienced psychiatrist it is evident that patients with schizophrenia are unable both t delusional programs and to realize these unrealistic intentions in the environment. I have named this dis "schizophrenic dysintentionality" (Mitterauer, 2005). But let us consider the elementary pathophysiologi of schizophrenia as outlined above. First of all, there is a break of information processing between the gl the neuronal system in tripartite synapses and perhaps also in the "orthogonal" oligodendrocyte-axonic this view, the term schizophrenia (split of consciousness) is appropriate. In other words: a patient with s permanently stressed by a world of intentions that cannot be mediated via tripartite synapses to the neu reality testing. Such considerations could be explanatory with concern to recent findings of abnormalities matter of the brain (Hof et al, 2003; Kubicki et al, 2005). Supposing that a patient is under permanent p realize his/her intentional programs generated in the panglial syncytium, then the normal apoptosis coul or mutations in astrocytes and in the oligodendrocyte-myelin system could be activated. The effect is a white matter as observed in the cited studies. Considering the loss of oligodendrocytes which are norma interconnected with astrocytes via gap junctions, the decay of oligodendrocytes must also destruct the p syncytium in the sense of an increasing loss of gap junctions. This loss of gap junctions may again destr of the panglial syncytium to generate intentional programs.

Many patients with schizophrenia become increasingly psychobiologically exhausted in the chronic cours which is called schizophrenic residuum. Of course, the frequently observed disorders in neuronal networ a role, but the destruction of the panglial syncytium leading to a dysintentionality per se, may be basica

for the negative view of life, as typically seen in the schizophrenic residuum. Most impressively, if the pr is severely affected, these patients are incapable of planning. Therefore, what they want is merely the s simple biological needs (eating, drinking, smoking, getting money to buy something, etc.). One could al the destruction of the panglial syncytium all kinds of destiny are broken down as well.

How could astrocytes react to this disaster? Reactive astrocytosis may be a compensatory attempt. Rea occurs prominently in response to all forms of CNS injury or disease. Recent studies point to the role of astrocytes in helping to limit tissue degeneration and preserve function after CNS injury (Sofroniew, 20(in schizophrenia? If one interprets the degeneration of the panglial syncytium caused by stress as a fun injury, then reactive astrocytosis may here exert the same mechanism. However, in schizophrenia astro only react to injuries of the neuronal system, but also attempt to generate a new astrocytic syncytium ir degeneration of the panglial syncytium. In this way the patient can generate intentional programs and k destiny" alive. This conjecture is experimentally testable, if one compares the degree of dysintentionalit the ability to produce intentions or plans – of schizophrenic patients with and without reactive astrocyto

Finally, reactive psychosis can be seen in the light of the Astrocentric Hypothesis (Robertson, 2002). Ac hypothesis, astrocytes represent the core cells in the brain that not only control the glial-neuronal intera determine the functions within the panglial syncytium. Therefore, astrocytes may be capable – at least - repairing dysfunctions in the panglial syncytium, as may be the case in schizophrenia.

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