Brain this may have adverse effects on neuronal functioning to (rigger hitherto (AMÍ). This inflammatory response may react the brain when it becomes systemic.

Mediators of inflammation like the pro-inflammatory cytokines can locally affect the areas. Considering the locations and selectivity of the damage, it is tempting to speculate that immune activation underlies initiation of neurodegenerative diseases. 

Evidence for inflammation in the brain after AMI came from immunocytochemical detection of ICAM-positive small vessels in certain areas, including the prefrontal cortex, the somatosensory cortex, and the cortical activation. Staining for albumin and IgG revealed that specifically around the ICAM-positive vessels there had been leakage of the BBB. probably caused by cytokine producing leukocytes adhering to the vessel wall. In order to assess whether the inflammation rather than the surgery resulted in leakage of the BBB, we injected TNFα in the tail vein. Compared to the myocardial infarction rats, after TNFα injections the ICAM-positive vessels and Albumin and IgG extravasation was observed in the same areas of the brain.

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The increased aAb level to QR (more than two-three times above the control value) was revealed in the blood serum of patients with epilepsy and paroxysmal activity. In the neurological patient's groups this effect was not seen. In majority (86 % ) of patients with epilepsy, revealed by PAT-test, demonstrated positive correlation with long-time of the disease, frequencies of attack and the type of paroxysms. The level of autoantibodies to QR epitopes could be new clinical index substantially facilitating unbiased diagnosis of epilepsy, additional to clinical, neurophysiological and other method of examination. The role of quisqualate receptor in the molecular mechanism of epilepsy is discussed.

The sigmoid Emax model does not correspond to a molecular model. The molecular mechanisms of compounds interacting with the GABA_{A} receptor complex are not yet fully unravelled. We investigated the regulation of gene expression of tyrosine hydroxylase, the rate limiting enzyme of dopamine synthesis. While tyrosine hydroxylase mRNA levels were not modified, protein levels were dramatically decreased (>90%) in the ventral midbrain of homoygote mice. This decrease is correlated with a decrease in dopamine levels (>90%) in the striatum. Immunohistochemistry with a tyrosine hydroxylase polyclonal antibody shows no difference in the number of dopamine neurons of the ventral midbrain in homoygote compared to wild type mice. Moreover, the decrease in tyrosine hydroxylase levels is less marked in the arcuato-olfactory tubercle than the striatum where dopamine transporter levels are normally higher. The dramatic extent of the down regulation documented here is only achieved in other animal models by destruction of dopamine neurons, which ultimately induce Parkinson-like syndromes. The mice lacking the dopamine transporter show a spontaneous increase in their locomotor activity despite the marked down regulation of tyrosine hydroxylase and dopamine levels strongly suggests that blockade of the dopamine transporter with highly selective antagonists could be beneficial in alleviating symptoms of Parkinson disease in humans.

Male Wistar rats received a myocardial infarction through ligation of two left posterior wall branches 2-14 days after surgery the rats were perfused. A second group of rats was treated intravenously with 1 μg human recombinant Tumor Necrosis factor alpha (TNFa). Results: Evidence for inflammation in the brain after AMI came from immunocytochemical detection of ICAM-positive small vessels in certain areas, including the prefrontal cortex, the somatosensory cortex, and the cortical activation. Staining for albumin and IgG revealed that specifically around the ICAM-positive vessels there had been leakage of the BBB, probably caused by cytokine producing leukocytes adhering to the vessel wall. In order to assess whether the inflammation rather than the surgery resulted in leakage of the BBB, we injected TNFα in the tail vein. Compared to the myocardial infarction rats, after TNFα injections the ICAM-positive vessels and Albumin and IgG extravasation was observed in the same areas of the brain. 

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