# Abstract (English version)

***Purpose:*** Based on recent studies, which have determinated overexpression of efflux transporter MRP2 in blood-brain barier of patients with refractory epilepsy, it has been suggested that this overexpression could contribute to the farmacoresistance to antiepileptic drugs. The aim of our study was to test substrate affinity of selected antiepileptic drugs. In particular, we focused on phenobarbital, phenytoin, ethosuximide, primidone, valproate, carbamazepin, clonazepam and lamotrigin. Furthermore, the inhibitory potency of these antiepileptic to MRP2 was examined.

***Methods:*** To study substrate affinity of tested antiepileptic to MRP2, transport experiments were performed in epithelial cell monolayers cultivated on mikroporous membrane filtres. Particulary, MDCKII cells transfected with cDNA of human MRP2 and, as a control, MDCKII-NeO cells where the expression of MRP2 is supressed, were used. For detection of inhibitory potency of the antiepileptic drugs to MRP2, accumulation assays were carried out on MDCKII-MRP2 and MDCKII-NeO cell lines using calcein as a known fluorescent substrate of MRP2.

***Results:*** The tested antiepileptic drugs are neither substrates nor inhibitors of MRP2.

***Conclusion:*** Our data do not support the hypothesis that the overexpression of MRP2 in blood-brain barrier of resistant epileptics is involved in refractory epilepsy. The tested antiepileptic also do not influence, by means of MRP2 inhibition, MRP2-directed transport of its substrates.