**Interaction of the MRP2 transporter with selected anti-epileptic drugs**

Epilepsy is a common neurological disorder affecting approximately 1-2% of the population. In most of the patiens with epilepsy, seizures are well-controlled with currently available anti-epileptic drugs (AEDs). It is estimated that 30% of the patients fail to achieve seizure termination despite carefully optimized drug treatment. Most of these patients with refractory epilepsy are resistant to several AEDs with different mechanisms of action, which suggests that resistance in epilepsy is a multifactorial and drug-nonspecific phenomenon. Based on experimental and clinical studies, two major theories have been put forward to explain the development of pharmacoresistance in epilepsy. The target hypothesis holds that changes in properties of drug targets for AEDs may result into their reduced drug sensitivity. The transporter hypothesis contends that the over-expression of efflux multidrug-transporters in the brain leads to impaired access of AEDs to their targets. The largest and the most important family of multidrug-transporters expressed in the brain, is the ABC (ATP-binding cassette) transporter family. One of the transporters in this family, recently under study, is the multidrug resistance-associated protein 2 (MRP2). The MRP2 transporter expressed in the apical membrane of polarized cells in organs of absorption and excretion, e.g. cells of the BBB (blood-brain barrier), has an important impact on pharmacokinetic parameters of many drugs, for example AEDs. The goal of this review is to summarize the interaction of anti-epileptic drugs with ABC transporters, especially the MRP2 transporter, based on well-documented *in vitro* and *in vivo* studies.