Description for the general public

All over the world, there are over 50 million people suffering from epilepsy and in need of treatment. In the group of patients receiving pharmacological treatment, some 20 – 40 % are classified as drug-resistant patients. The ketogenic diet makes one of the most effective therapeutic approaches in patients with drug-resistant epilepsy. Despite a long history of applying the ketogenic diet in the treatment of epilepsy, the mechanism of action of the ketogenic diet is not fully understood yet. In the studies regarding mechanism of anticonvulsant action of fatty acid (FA) contained in KD, we observed that FA causes an increase in the brain concentration of tryptophan (TRP) and its metabolites including kynurenic acid (KYNA) and kynurenine (KYN). In addition, we observed that the administration of FA exerts acute anticonvulsant effects which can be abolished by blocking the transport of TRP into the brain (Maciejak 2016). The results of our latest research permitting a hypothesis regarding the mechanism of KD action as based on modifications of TRP metabolism and kynurenine pathway as well as on the subsequent significant biochemical and oxy-reduction changes causing reduced neuron excitability. Finding new binding sites for the substances regulating those processes might increase the likelihood of replacing KD with pharmacological action. An optimized composition of the ketogenic diet and/or administering pharmacological substances would allow us to make use of the efficacy of KD without the necessity to keep its rigorous dietetic regime.

Assuming an optimal course of the research, this would prospectively allow to suggest potential binding sites for pharmacological action aimed at improved efficacy and, more significantly, improved quality of life for patients using the ketogenic diet by replacing the rigorous dietary regime with a substance inducing a similar metabolic effect.