**Na,K-ATPase Activity in the Hippocampus of the Pilocarpine-Induced Rat Model of Epilepsy Treated with Benzylidene Digoxin 15 (BD15)**

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Na,K-ATPase (NKA) is an essential enzyme in maintaining the sodium and potassium ion gradient across the plasma membrane of neuronal cells, playing a crucial role in regulating neuronal excitability and ionic homeostasis. Dysfunctions in NKA have been associated with various neurological conditions, including epilepsy. Studies indicate that genetic mutations affecting Na,K-ATPase functionality can lead to imbalances in ion concentrations, resulting in neuronal hyperexcitability and a predisposition to epileptic seizures. Thus, Na,K-ATPase emerges as a key point in understanding the underlying pathophysiological mechanisms of epilepsy and a potential therapeutic target for developing new treatments. A semi-synthetic analogue to digoxin, benzylidene digoxin 15 (BD15), has shown promise as a neuroprotective drug. Therefore, this study aims to evaluate the effect of BD-15 on NKA activity in an experimental model of temporal lobe epilepsy. Male Wistar rats were induced with a high dose of pilocarpine (300 mg/kg; i.p.) and assessed for status epilepticus (SE). The animals were then divided into groups: CTR (control); CTR+BD15 (control with BD15); NSE (no SE); NSE+BD15 (no SE with BD15); SE (with SE) and SE+BD15 (with SE with BD15). The animals received BD15 (100 µg/kg; i.p.) for 3 days, while the others received 0.9% saline. On the seventh day, the animals were euthanized, and the hippocampus was collected. A homogenate was prepared from the sample, and the total NKA activity, as well as the activities of the alpha 1, 2, and 3 isoforms, were measured. The results show that the NSE and SE groups had reduced total activity and reduced activities of the alpha 2 and 3 isoforms, while the groups treated with BD15 had values similar to the control group. No difference was observed in the activity of alpha 1 among the groups. Thus, we observed that the experimental model of Temporal Lobe Epilepsy showed a normalization of NKA activity with BD15 treatment.