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Reduced Activity of Red Cell Na⁺ K⁺ ATPase and Ca²⁺ ATPase in Patients with Idiopathic Generalized Epilepsy

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ABSTRACT Evidence is now emerging that certain epilepsies may be a family of channelopathies with defects involving mutations in the Na⁺, K⁺, or Ca²⁺ channels whose activities are related to their voltage dependent conditions, or defects in the membrane-bound enzymes Na⁺ K⁺ ATPase and Ca²⁺ ATPase that regulate the transport of ions across the cell membrane. In this study we have tried to determine the role of the membrane bound enzymes, sodium-potassium and calcium ATPase in the aetiopathogenesis of idiopathic generalized epilepsies (IGE). We studied 143 cases for sodium -potassium ATPase and 109 cases for calcium ATPase enzymes in comparison to 80 matched controls. Citrated blood samples were collected from these cases and red cells separated from the plasma. Erythrocyte membranes were prepared and tested for activity of both the enzymes. We found significantly lowered activities of both the enzymes in IGE cases as compared to control samples. The familial cases (cases with positive family history) had significantly lower activities of the enzymes than non-familial cases, as did females as compared to males. Our data indicates that genetic defects in the active transport mechanisms like Na⁺ K⁺ ATPase and Ca²⁺ ATPase enzymes are associated with human idiopathic epilepsy. These findings add to the growing list of channelopathies in humans and suggest that drugs that directly or indirectly modulate K⁺, Na⁺ and Ca²⁺ ion channels will be helpful in the treatment of seizure disorders.

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