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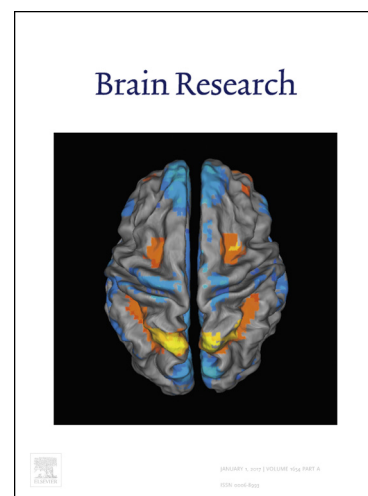
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Oxidative stress and Na,K-ATPase activity differential regulation in brainstem and forebrain of Wistar Audiogenic Rats may lead to increased seizure susceptibility

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ABSTRACT

The Wistar Audiogenic Rat (WAR) is a well-characterized seizure-prone, inbred rodent strain that, when acutely stimulated with high-intensity sounds, develops brainstem-dependent tonic-clonic seizures that can evolve to limbic-like, myoclonic (forebrain) seizures when the acoustic stimuli are presented chronically (audiogenic kindling). In order to investigate possible mechanisms underlying WAR susceptibility to seizures, we evaluated Na,K-ATPase activity, Ca-ATPase activity, Mg-ATPase activity, lipid membrane composition and oxidative stress markers in whole forebrain and whole brainstem samples of naïve WAR, as compared to samples from control Wistar rats. We also evaluated the expression levels of $\alpha 1$ and $\alpha 3$ isoforms of Na,K-ATPase in forebrain samples. We observed increased Na,K-ATPase activity in forebrain samples and increased oxidative stress markers (lipid peroxidation, glutathione peroxidase and superoxide dismutase) in brainstem samples of WAR. The Ca-ATPase activity, Mg-ATPase activity, lipid membrane composition and expression levels of $\alpha 1$ and $\alpha 3$ isoforms of Na,K-ATPase were unaltered. In view of previous data showing that the membrane potentials from naïve WAR's neurons are less negative than that from neurons from Wistar rats, we suggest that Na,K-ATPase increased activity might be involved in a compensatory mechanism necessary to maintain WAR's brains normal activity. Additionally, ongoing oxidative stress in the brainstem could bring Na,K-ATPase activity back to normal levels, which may explain why WAR's present increased susceptibility to seizures triggered by high-intensity sound stimulation.

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