NEURONAL EXCITABILITY CHANGES DURING LPS-INDUCED HYPERTHERMIC RESPONSE IN BIOTELEMETERED RATS *

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Background- The neuronal excitability time-dependently changes after lipopolysaccharide (LPS) administration (1). A proconvulsant activity against pentylenetetrazol (PTZ)-induced seizures can be observed on the relatively early time period (such as 4 h) after LPS challenge. Meanwhile, a hypothermic response also occurs in this time period (2). Thus, we hypothesized that the reduction in body temperature (Tb) may lead to the neuronal hyperexcitability and we decided to evaluate the neuronal excitability changes at the various phases of LPS-induced hypothermia in rats. Procedure- Tb of adult male albino Wistar rats was recorded by telemetry (Mini-Mitter). E. coli O111:B4 LPS was injected (250 microgram/kg, ip). Seizures were induced by PTZ (60 mg/kg, ip) at the initial phase, at the nadir and at the end of the hypothermic response. The incidence and the latency of generalized tonic-clonic convulsions were noted. On the other experimental set-up, Tb was recorded telemetrically together with EEG (DSI, Transoma). EEG power spectra and spike-wave activity were analyzed by Dataquest (ART; v 3.1) and NeuroScore seizure module (version 1.1), respectively. Results- The latency of seizures was reduced at the initial phase, but a clear anticonvulsive activity was observed at the end of the hypothermic period. The seizure parameters did not change at the nadir. There was no EEG spike activity on the either phase of the response. Meanwhile, the power of 1-4 Hz delta band of the EEG spectra decreased at the initial phase of the hypothermia. Conclusions- The data show that spike activity is not facilitated by LPS treatment in rats. But, proconvulsant and anticonvulsant activities occur in a sequence depending on the phases of LPS-induced hypothermic response. The EEG power spectra also change. It seems that these effects may not be attributed merely to the reduction of Tb. It is more conceivable to suggest that the pathophysiological mechanisms, which account for the hypothermia, may also be responsible for the neuronal excitability changes.


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