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LncRNA HOTAIR Modulates Lipopolysaccharide-Induced Inflammatory Injury in Neuronal Cell Line HT-22

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ABSTRACT Epilepsy was recently emerged as a prevalent neurological disorder of the central nervous system (CNS). the In this work, the researchers aimed to explore the functional character of one of non-coding RNA (lncRNA) Hox transcript antisense RNA (HOTAIR) in hippocampal HT-22 after lipopolysaccharide (LPS) modulation. An in vitro inflammatory cell-based model was constructed by LPS treatment and inflammatory cell injury was monitored through the changes of cell viability, cell apoptosis, and levels of inflammatory cytokines. HOTAIR expression was inhibited via cell transfection, and the impact of HOTAIR depletion on LPS modulated cell inflammatory injury, and key kinases of NF-κB and MEK/ERK pathways were investigated after LPS stimulation by the qRT-PCR method. Results presented that LPS treatment led to the upregulation of HOTAIR in HT-22 cells, and LPS-induced cell inflammatory injury was reduced by HOTAIR knockdown. Intriguingly, HOTAIR depletion suppressed the phosphorylated levels of crucial kinases of both NF-κB and MEK/ERK pathways. Hence, this study concluded that LPS upregulated HOTAIR, and HOTAIR could modulate the LPS-induced cell inflammatory injury via NF-κB and MEK/ERK pathways.