A Novel Identification of the Epigenetic Enzyme JMJD1a in Neuroinflammation

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Neuroinflammation in the hippocampus and hypothalamus constitutes the common pathology underlying various neurodegenerative diseases and metabolic syndrome, respectively. Environmental inducers of neuroinflammation, including infections, toxin exposure, and mechanical injury, may change brain epigenetic regulation of gene expression, which can lead to alterations in neural activity and metabolic imbalances. Thus, I hypothesize that epigenetics, functioning at the gene-environment interface, may play a role in the development of diseases with neuroinflammatory-related pathologies. In this research, high-grade acute and chronic neuroinflammatory phenotypes were successfully generated in C57BL/6J mice through the intraperitoneal administration of lipopolysaccharide (LPS). After screening epigenetic enzymes in the hippocampus and hypothalamus through qPCR, I further determined the protein levels of the key epigenetic enzymes through Western Blotting. The results indicated that LPS-induced neuroinflammation was associated with overall elevated hippocampal and hypothalamic gene and protein expression levels of Jumonji C domain-containing protein 1a (JMJD1A), a histone demethylase. Furthermore, I discovered that high-fat diet-induced low-grade neuroinflammation in mice is also associated with marginally elevated hippocampal gene and protein expression levels of JMJD1a. These results unravel, for the first time, that hippocampal and hypothalamic JMJD1a is responsive to neuroinflammation and could play an important epigenetic regulatory function in the inflammatory pathways. The discovery of JMJD1a could pave a new avenue for combating neuroinflammatory diseases and advance our knowledge in neuroinflammatory processes and the related mechanisms.

Awards Won:

Fourth Award of $500

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