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Poster Presentation

Evaluating the Role of Histone Hyper Acetylation in Induction of Neuroinflammation

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Abstract

Microglia is the effector cell of the innate immune system in central nervous system (CNS). These cells mediate inflammatory responses in injuries. Besides external factors, microglial function is also controlled by internal factors, including epigenetic regulations. Mechanisms of epigenetic regulation mainly consist of DNA methylation, histone modifications and use of non-coding RNAs. Recent studies have demonstrated that these epigenetic processes can alter the function of microglia and thus, adjust neuroinflammation. Neuroinflammation is believed to play a significant role in development of numerous neurological disorders, including Multiple Sclerosis (MS), which is the most prevalent chronic inflammatory disease of CNS. Therefore, it has been hypothesized that the aforementioned epigenetic processes could act as a potential therapeutic target for neuroinflammatory diseases and many studies have been performed in this field. Among various histone modifications, histone acetylation is the most studied subjet. Previous studies demonstrate that histone hyper acetylation in various tissues can contribute to inflammation. Although no studies have specifically evaluated the role of histone hyper acetylation in inducing neuroinflammation so far, but multiple studies have acknowledged the beneficial use of histone deacetylase in limiting neuroinflammation. Thus, it can be concluded that histone hyper acetylation is associated with neuroinflammation. We believe that more research is needed to assess the relationship between histone hyper acetylation and neuroinflammation, and to investigate whether or not hyper acetylation in microglia can induce inflammatory response in CNS. Moreover, we suggest evaluating the possibility of epigenetic transgenerational inheritance of neuroinflammation through histone hyper acetylation, since most of the previous studies in this field have focused on epigenetic inheritance in neuronal behaviors through miRNAs and DNA methylation.

Keywords: Epigenetics, Histone, Histone acetylation, Neuroinflammation, Inflammation

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