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H2A.Z in Neuron: Implications in Pain-Related Social Defect

Xian Wang*,Δ

SUMMARY Cumulating evidence indicated that chronic pain-associated cellular and molecular changes play an essential role in contributing to the development of social defect symptoms, and more recent findings implicated the critical role of epigenetic mechanisms in chronic pain-related sensitization. Histone variant exchange, in which canonical histones are replaced with their variant counterparts, is an entire branch of epigenetics that has received limited attention in the brain and has never, to our knowledge, been studied in relation to pain-related social defect. Here we hypothesize that H2A.Z, a variant of histone H2A, is actively involved in the regulation of chronic pain related social defect symptoms, probably through downstream effects on gene expression. We hope the histone variant H2A.Z regulation may contribute to the molecular basis of cognitive function and serve as a potential therapeutic target for associated social defect.■

*: Department of Anesthesiology and Intensive Care Medicine, Affiliated Nanjing Maternity and Child Health Care Hospital, Nanjing Medical University, Nanjing, Jiangsu, China

Δ: Correspondence to: No.123, Tianfei Xiang, Mochou Rd., Nanjing 210004, Jiangsu, China. Tel: (+)86-(0)25-5222-6112, E-mail: wangxian2002@126.com

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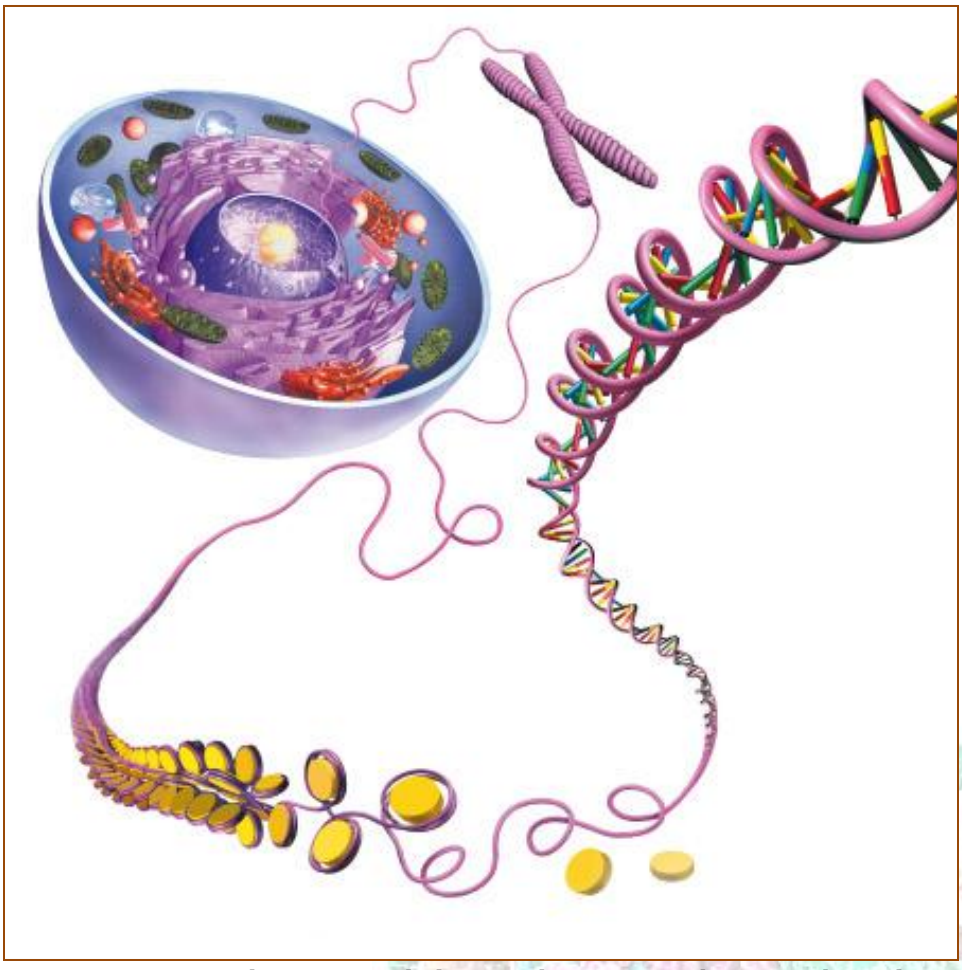
SOCIAL DEFECT underlies a combination of series of severe psychiatric conditions such as anxiety, depression, autism, schizophrenia, or suicidal tendency (1). At the same time, a considerable proportion of patients with chronic pain have been observed to have an increased risk of developing social defected symptoms (2). Considering the high comorbidity rate of chronic pain and social defect, it is important to explore the underlying etiological and pathophysiological mechanisms, and verifies the therapeutic efficacy of possible target interventions on the comorbidity as well.

Numerous studies have suggested the involvement of epigenetic mechanisms in the development of chronic pain and diverse neurological diseases underlying social defect symptoms. Tran and colleagues found that epigenetic programming in the amygdala, specifically histone modifications, is important in the maintenance of chronic anxiety and pain (3). Sampathkumar et al (4) outlined the importance of epigenetic modifications in neurodevelopmental disorders, especially autism spectrum disorders.



Epigenetic mechanisms include DNA methylation, histone modifications, nucleosome repositioning, higher order chromatin remodeling, non-coding RNAs, and RNA and DNA editing. Menke and colleagues suggested that current psychopharmacologic drugs including antidepressants, antipsychotics and mood stabilizers may exert some of their effects by inducing epigenetic changes (5). Epigenetic processing had been suggested as prospective molecular indicators of the biological consequences of stress and chronic pain (6).

Recently, H2A.Z, a universally conserved variant of histone H2A, an entire branch of epigenetics, has been demonstrated in the regulation of series of cognitive disorders. H2A.Z is found in nucleosomes within the promoters of most genes from yeast to plants to humans, and plays an essential role in transcriptional memory. Zovkic and coresearchers provided evidence that histone variant exchange was a novel mechanism contributing to the molecular basis of cognitive function and implicated H2A.Z as a potential therapeutic target for memory disorders (7). Besides, Maze et al (8) suggested histone regulation appears to have important roles in both the developing and adult CNS, and seems to be critical to many aspects of neural plasticity that directly influence the establish ment of complex behavioral phenotypes. Considering the role of histone variant H2A.Z in cognitive function, we hope it may serve as a potential therapeutic target for social defect symptoms.

Combining our previously established behavioral model of chronic pain-included social defect (9), in the future, we aimed to originally characterize the mechanisms underlying H2A.Z and social defect symptoms in patients with chronic pain and determine whether H2A.Z may serve as an effective therapeutic target for pain-related social defect symptoms.■

Conflict of Interests

None

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