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Over-activation of G9a induced by oxidative stress disturbs the expression of RE-1-containing neuronal specific genes and leads to degeneration in human SH-SY5Y neuroblastoma cells

Goang Won Cho
Chosun University, Republic of Korea

Neuronal degeneration induced by chronic oxidative stress is one of several etiological causes of neurodegenerative diseases. In this study, we investigated the functional role of histone methyltransferase G9a in oxidative stress-induced degeneration in human SH-SY5Y neuroblastoma cells. Using the MTT assay and immunoblot analysis, the viabilities of H_2O_2-treated cells decreased significantly; however, treatment with the G9a inhibitor BIX01294 partially attenuated this effect. The expression of neuronal specific genes also decreased in H_2O_2-treated cells but recovered with G9a inhibition. H_2O_2-treated cells showed high levels of H3K9me2 (histone H3 dimethylated at the lysine 9 residue), which is produced by G9a activation, and BIX01294 treatment lessened the over-activation of G9a. The expression of repressor element-1 (RE-1)-containing neuronal specific genes increased in response to BIX01294 treatment as confirmed by a chromatin immunoprecipitation (ChIP) assay with H3K9me2 antibody. The differentiation rates of SH-SY5Y cells also recovered with the inhibition of G9a in H_2O_2-treated cells, which was seen by immunoblot analysis of neuronal specific proteins. These results demonstrate that oxidative stress induces the over-activation of G9a, disturbs the expression of neuronal specific genes, and progressively mediates neuronal cell death. Moreover, a G9a inhibitor can lessen G9a over-action and prevent neuronal damage. G9a inhibition may therefore contribute to the prevention of oxidative stress-induced neurodegeneration.

Biography

Goang Won Cho has completed his PhD program from Hanyang University (Seoul, Korea) and Postdoctoral studies from National Institutes of Health (NIH, USA) and Duke University (USA). He is the Professor of Chosun University. He has published dozens of SCI papers.

gwcho@Chosun.ac.kr

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