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Excessive apoptosis and disordered autophagy flux contribute to the neurotoxicity induced by high iodine in Sprague-Dawley rat

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Abstract

In recent years, the detrimental effects of high iodine on intelligence are gaining tons of attention, but the relationship between high iodine and neurotoxicity is controversial. This study aimed to explore whether high iodine intake may impair intelligence and the roles of apoptosis and autophagy in high iodine-induced neurotoxicity. The results showed that high iodine exposure reduced brain coefficient and intelligence of rats, and caused histopathological abnormalities in hippocampus. Moreover, high iodine increased hippocampal apoptosis, as confirmed by elevation of apoptotic proteins and TUNEL-positive incidence. Further study showed that high iodine impaired mitochondrial ultrastructure and caused elevation of Bax, cytochrome c and decline of Bcl2, indicating the participation of mitochondrial apoptotic pathway. Simultaneously, high iodine also increased the number of autophagosomes. Intriguingly, the expression of autophagosomes formation protein Atg7, Beclin1 and autophagic substrate p62 were elevated, suggesting that the accumulated autophagosomes is not only due to the enhancement of formation but also the decline of clearance. These, together with the numerous damaged organelles observed in hippocampal ultrastructure, reveal the crucial role of disordered autophagy flux in high iodine-elicited neurotoxicity. Collectively, these findings suggest that excessive apoptosis and disordered autophagy flux contribute to high iodine-elicited neurotoxicity.

Keywords: Apoptosis; Autophagy flux; Iodine; Neurotoxicity; Rats.

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