

CHANGED MUSCARINIC ACETYLCHOLINE RECEPTORS AND THE LEVEL OF OXIDATIVE STRESS IN THE BRAINS OF THE RATS WITH CHRONIC FLUOROSIS

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SUMMARY: Cross-sectional studies were conducted to explore the relationships between high fluoride exposure from drinking water and the development of essential hypertension and carotid artery atherosclerosis. The subjects were adults who were randomly recruited from eight villages in an endemic area of fluorosis in Zhaozhou County, Heilongjiang Province, China. The range of fluoride concentrations of drinking water were: normal group (less than 1.20 mg/L), mild group (1.21–2.00 mg/L), moderate group (2.01–3.00 mg/L), and heavy group (more than 3.01 mg/L). The results of the study showed that as the water fluoride concentrations increased, the prevalence of essential hypertension and carotid artery atherosclerosis also increased significantly, and that high water fluoride concentrations were closely associated with essential hypertension (odds ratio: heavy=2.84) and carotid artery atherosclerosis (odds ratios: mild=1.92, moderate=2.01, and heavy=2.38). In addition, significant differences were seen in the plasma endothelin-1 (ET-1) levels in the subjects in the different groups ($p < 0.0001$), and the plasma ET-1 level was closely related to the occurrence of essential hypertension. At the same time, it was found that elevated intercellular cell adhesion molecule-1 (ICAM-1) and reduced glutathione peroxidases (GPx) were associated with carotid artery atherosclerosis. Thus, the findings suggested that the possible mechanism of this positive relationship between excess fluoride exposure from drinking water and carotid artery atherosclerosis or essential hypertension might be that the excess intake of fluoride induced oxidative stress with stimulation of the production of ICAM-1 and ET-1 and resulting in an inflammatory reaction and endothelial activation.

Key words: Brain; Chronic fluorosis; GSH-Px; Learning and memory; mAChRs; Muscarinic acetylcholine receptors; Oxidative stress; Rats; SOD.

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