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Association of the genes for tumor necrosis factor-α and myelin basic protein with delayed encephalopathy after acute carbon monoxide poisoning

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ABSTRACT. There is structural damage to myelin and secondary immune injury in the development of delayed encephalopathy after acute carbon monoxide (CO) poisoning (DEACMP). In order to assess the role of genetic factors in this mechanism, we studied the association between tumor necrosis factor-α308 (TNF-α308) and myelin basic protein (MBP) 5'-side tetranucleotide repetitive sequence (TGGA) n gene polymorphism and DEACMP. We selected 109 DEACMP patients from the Han population in the Northern Henan Province as the case group, and 115 patients without delayed encephalopathy (called the acute CO poisoning group or the control group). There were no significant differences in TNF-α308 and MBP 5'-side TGGA n genotype

distribution and allele frequency between the DEACMP group and the acute CO poisoning group (all P > 0.05). When the population was stratified by gender, only the MBP 5'-side TGGA n allele frequency was significantly different, and the frequency of allele L in the DEACMP group was significantly higher than that of the acute CO poisoning group in males (χ^2 = 4.089, P = 0.043, odds ratio = 2.103, 95% confidence interval = 1.014-4.363). The results showed that there was association between MBP 5'-side TGGA n gene polymorphism and DEACMP, and that allele L could increase the risk of occurrence in male patients with DEACMP. DEACMP may be the result of interaction of environmental and genetic factors.

Key words: Carbon monoxide poisoning; Encephalopathy; Tumor necrosis factor-α; Myelin basic protein; Gene polymorphism