Meta-analysis of the relationship between slow acetylation of N-acetyl transferase 2 and the risk of bladder cancer

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ABSTRACT. The incidence of bladder cancer is closely associated with exposure to aromatic amines, that can cause cancer only after metabolic activation regulated by N-acetyl transferase 1 and 2 (NAT1 and NAT2). Many studies have indicated that slow acetylation of NAT2 increases the risk of bladder cancer. The major risk factor is tobacco smoke; however, some studies have failed to prove this. This study attempted to explore the correlation between NAT2 slow acetylation and bladder cancer risk through a meta-analysis of published case-control studies. Studies detecting *NAT2* gene status in bladder cancer patients and healthy controls were retrieved

from PubMed, Cochrane, EMchrane, CBM, and CNKI. We retrieved the data of cited articles and publications to identify and compare NAT2 gene in bladder cancer patients and healthy controls. The variables within and between the studies were also considered. The META module in the Stata v.6.0 software was used for data analysis. Twenty independent studies were enrolled in our meta-analysis according to the inclusion and exclusion criteria. Individual differences in the bladder cancer susceptibility were, in part, attributed to the effect of carcinogens. The merged odds ratio of the effect of slow acetylation on bladder cancer was 1.31 (95% confidence interval = 1.11-1.55). In conclusion, NAT2 slow acetylation state was associated with bladder cancer risk, and was shown to modestly increase the risk of bladder cancer.

Key words: NAT2; NAT2 slow acetylation; Bladder cancer

