

## THE EFFECT OF TREATMENT AND MECHANISM OF ASTRAGALOSIDE ON CHRONIC VIRAL MYOCARDITIS WITH COXSACKIEVIRUS B<sub>3</sub> IN BALB/C MICE

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**Objective:** To Observe the effect of the treatment and mechanism of cardiac apoptosis of astragaloside on chronic viral myocarditis with CVB3 in Balb/c mice.

**Methods:** Four-week-old Balb/c mice were randomly divided into three groups as follows: astragaloside treatment group, model of viral cardiomyopathy group, and normal control group. The model group and control group were fed with drinking water while astragaloside treatment group with drinking water containing astragaloside at concentration of 300mg/L for 3 months. Survival rates were determined, myocardial histopathology, Collagen volume fraction (CVF) and apoptosis of heart tissue, levels of Bax, Bcl-2 and CVB<sub>3</sub>-RNA levels (realtime RT-PCR) were detected on 3 months later respectively.

**Results:** The survival rate on 3 months was significantly improved in mice treated with Astragaloside in treatment group than that of model group ( $p < 0.05$ ). Astragaloside treatment also significantly attenuated histological myocardial lesion, reduced the myocardial CVF and apoptosis, decreased myocardial CVB<sub>3</sub>-RNA levels ( $P < 0.01$  or  $p < 0.05$ , respectively). The Astragaloside significantly induced the expression levels of Bcl-2 protein ( $p < 0.05$ ) and mRNA ( $p < 0.05$ ), reduced the expression level of Bax protein ( $p < 0.05$ ) and mRNA ( $0.63 \pm 0.13$  vs  $0.79 \pm 0.12$ ,  $p < 0.05$ ) than that in viral cardiomyopathy group. respectively.

**Conclusion:** Astragaloside is a potent agent with a highly significant effect on chronic viral myocarditis in the Balb/c murine with CVB<sub>3</sub>. and could reduce the apoptosis of viral myocarditis. Which mechanism may be partly up-regulated expression of Bcl-2 and down-regulated expression of gene Bax.