MicroRNA-122 is involved in oxidative stress in isoniazid-induced liver injury in mice

L. Song¹, Z.R. Zhang¹, J.L. Zhang¹, X.B. Zhu¹, L. He¹, Z. Shi¹, L. Gao¹, Y. Li¹, B. Hu¹ and F.M. Feng¹

¹Hebei Province Key Laboratory of Occupational Health and Safety for Coal Industry, School of Public Health, North China University of Science and Technology, Tangshan, China
²Center for Disease control and Prevention of Bayannur, Bayannur, China

Corresponding author: F.M. Feng
E-mail: fm_feng@sina.com

Received May 6, 2015
Accepted July 24, 2015
Published October 26, 2015
DOI http://dx.doi.org/10.4238/2015.October.26.22

ABSTRACT. Many studies have shown that the pathogenesis of liver injury includes oxidative stress. MicroRNA-122 may be a marker for the early diagnosis of drug-induced liver injury. However, the relationship between microRNA-122 and oxidative stress in anti-tuberculosis drug-induced liver injury remains unknown. We measured changes in tissue microRNA-122 levels and indices of oxidative stress during liver injury in mice after administration of isoniazid, a first-line anti-tuberculosis drug. We quantified microRNA-122 expression and indices of oxidative stress at 7 time points, including 1, 3, and 5 days and 1, 2, 3, and 4 weeks. The tissue microRNA-122 levels and oxidative stress significantly changed at 3 and 5 days, suggesting that isoniazid-induced liver injury reduces oxidative stress and microRNA-122 expression compared to in the control group (P < 0.05). Notably, over the time course of isoniazid-induced liver injury, mitochondrial ribosome protein S11 gene, the target of microRNA-122, began to change at 5 days (P < 0.05). The tissue microRNA-122 profile may affect oxidative stress by regulating mitochondrial ribosome protein...
S11 gene during isoniazid-induced liver injury, which may contribute to the response mechanisms of microRNA-122 and oxidative stress.

**Key words:** Anti-tuberculosis drug-induced liver injury; Isoniazid; MicroRNA-122; Mitochondrion; Oxidative stress