GMR Genetics and Molecular Research
Online Journal
ISSN:1676-5680

Effects of acrylonitrile on lymphocyte lipid rafts and RAS/RAF/MAPK/ERK signaling pathways

X.J. Li, B. Li*, J.S. Huang, J.M. Shi, P. Wang, W. Fan and Y.L. Zhou*

Department of Occupational Health, Jinshan Hospital, Fudan University, Shanghai, China

*These authors contributed equally to this study.
Corresponding authors: Y.L. Zhou / B. Li
E-mail: zhouyl2000@vip.sina.com / libing22@126.com

Genet. Mol. Res. 13 (3): 7747-7756 (2014)
Received May 7, 2014
Accepted September 11, 2014
Published September 26, 2014
DOI http://dx.doi.org/10.4238/2014.September.26.12

ABSTRACT. Acrylonitrile (ACN) is a widely used chemical in the production of plastics, resins, nitriles, acrylic fibers, and synthetic rubber. Previous epidemiological investigations and animal studies have confirmed that ACN affects the lymphocytes and spleen. However, the immune toxicity mechanism is unknown. Lipid rafts are cell membrane structures that are rich in cholesterol and involved in cell signal transduction. The B cell lymophoma-10 (Bcl10) protein is a joint protein that is important in lymphocyte development and signal pathways. This study was conducted to examine the *in vitro* effects of ACN. We separated lipid rafts, and analyzed Bcl10 protein and caveolin. Western blotting was used to detect mitogen-activated protein kinase (MAPK) and phosphorylated MAPK levels. The results indicated that with increasing ACN concentration, the total amount of Bcl10 remained stable, but was concentrated mainly in part 4 to part 11 in electrophoretic band district which is high density in gradient centrifugation. Caveolin-1 was evaluated as a lipid raft marker protein; caveolin-1 content and position were relatively unchanged. Western blotting showed that in a certain range, MAPK protein was secreted at

a higher level. At some ACN exposure levels, MAPK protein secretion was significantly decreased compared to the control group ($P < 0.05$). These results indicate that ACN can cause immune toxicity by damaging lipid raft structures, causing Bcl10 protein and lipid raft separation and restraining Ras-Raf-MAPK-extracellular signal-regulated kinase signaling pathways.

Key words: Acrylonitrile; Bcl10 protein; Caveolin-1; Lipid rafts; MAPK

