conferenceseries.com

J Vet Sci Technol 2018, Volume 9
DOI: 10.4172/2157-7579-C2-040

11th International

VETERINARY CONGRESS

July 02-03, 2018 Berlin, Germany

Cadmium induces Ca^{2+} mediated, calpain-1/caspase-3-dependent apoptosis in primary cultured rat proximal tubular cells

Hong Wang
Nanjing Agricultural University, China

Calcium, as a ubiquitous second messenger, governs a large array of cellular processes and is necessary for cell survival. More recently, it was observed that the cytosolic calcium ($[Ca^{2+}]_c$) elevation could induce apoptosis in primary cultured rat Proximal Tubular (rPT) cells exposed to Cd, but the concrete mechanism is still unclear. This study was designed to investigate the signal pathway involved in $[Ca^{2+}]_c$ elevation-mediated apoptosis. The results confirmed the elevation of $[Ca^{2+}]_c$ by confocal microscopy and enhancement of the apoptosis by Hoechst 33258 staining and flow cytometer when rPT cells were exposed to Cd for 12 h. Then we demonstrated that Cd enhanced the protein levels of active calpain-1 and caspase-3 in rPT cells. Pretreatment with the intracellular calcium chelator BAPTA-AM markedly blocked the up-regulation of active calpain-1 and caspase-3 and inhibited the apoptosis induced by Cd. Further, rPT cells were pretreated with a cell-permeable selective calpain-1 inhibitor PD150606 and caspase-3 inhibitor Ac-DEVD-CHO, respectively. PD150606 significantly attenuated the up-regulation of active caspase-3 and the apoptosis induced by Cd. As expected, inhibition of active caspase-3 by Ac-DEVD-CHO decreased the apoptosis induced by Cd. Taken together, it could be concluded that $[Ca^{2+}]_c$ elevation did act as a pro-apoptotic signal in Cd-induced cytotoxicity of rPT cells, triggered calpain-1 and caspase-3 activation in turn, and induced apoptosis of rPT cells.

2016207032@njau.edu.cn