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Identification of the proteins related to HQ-induced cytotoxicity by proteomic analysis

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Hydroquinone (HQ), one of the most important metabolites derived from benzene, is known to be associated with acute myelogenous leukemia risk; however, its carcinogenic mechanism remains unclear. Recently, we reported that ROS participated HQ-induced cytotoxicity in TK6 cells. To explore the molecular mechanism of cell response induced by HQ in TK6 cells. We treated TK6 cells with HQ 20μmol/L for 24h, to further identify the proteins related to HQ induced cytotoxicity by using the techniques of 2D electrophoresis and MALDI-TOF–MS/MS. After HQ treatment, cells were lysed in lysis solution, then the supernatant were collected and . The total cellular proteins were separated using two-dimensional gel electrophoresis and visualized by colloidal comassie blue staining. Digital image were analyzed using ImagerMaster 2Dplatinum 5.0 software. The deferentially expressed protein spots were picked and digested in gel then identified by MALDI-TOF–MS/MS. Among the 31 differential proteins identified, 21 Down-regulated proteins identified in the TK cells induced by HQ were found to be mainly involved in cell proliferation and migration, DNA replication, tumor protein and cytoskeleton protein , including purine biosynthesis protein,Ras GTPase-activating protein-binding protein 1, Keratin. Meanwhile, 8 proteins involved in some oxidative and ubiquitination were up-regulated in the TK6 cells induced by HQ , including NADH-ubiquinone oxidoreductase, HSP70 and Ubiquitin-conjugating enzyme E2N. And we applied western-blot to validated HSP70 and Ubiquitin-conjugating enzyme E2N in TK6 cells treated by 0,5,10,20μmol/L HQ treated for 24h. The results showed that the protein expression level of HSP70 and Ubiquitin-conjugating enzyme E2N were up-regulated along with the diferent concentration of HQ treatment. Our research indicated that oxidative stress, RAS pathway and ubiquitination may be involved in HQ induced acute cytotoxicity in TK6 cells.

Biography

Yan Sha received her Ph.D in toxicology from Sun Yat-sen University at Guangzhou, China. She completed her visitor schloar fellowship in Oregon Institute of Occupational Health Sciences, Oregon Health and Science University, Portland, Oregon. Dr Sha is a researcher from Shenzhen Prevention and Treatment Center for Occupational Diseases, her research focused on both DNA repair and molecular mutagenesis induced by workplace chemicals. She has published about 15 papers in reputed journals.

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