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Abstract
The incidence of bladder cancer in southwest coast of Taiwan, where blackfoot disease is endemic, is unusually high. Epidemiological studies have established a link between arsenic and bladder cancer, but the process transforming normal urothelium to cancer cells remains to be elucidated. Previous studies indicate that arsenic may exert its effect by inducing expression of proteins and stimulating the protein kinases and transcription factors. However, a global survey of arsenic-associated profile at the protein level has not been conducted. The overall goal of this proposal is to utilize two-dimensional gel electrophoresis and mass spectrometry to profile the protein expression induced by low-dosage arsenic in immortalized E6 cells.
In this study, the arsenic treated immortalized urothelial cells (E6) was used to evaluate the carcinogenesis of the bladder cancer induced by arsenic. The E6 cells were treated with 0.1 ppm for 1 week, 0.2 ppm for 1 week, 0.3 ppm for 1 week and 0.4 ppm for 1 week with total of two months, three months, four months and six months. In addition, that was treated for three months with 0.4 ppm, 0.8ppm and 1.2ppm, respectively. By comparison of the proteome maps of arsenic-treated E6 cells with those of E6 cells, ten down-regulated and eleven up-regulated protein spots were found. The identities of ten down-regulated proteins were macrophage capping protein, catechol-o-methyltransferase, aldehyde dehydrogenase dimeric NADP- preferring, elongation factor 1 gamma, glycyl tRNA synthetase, Ran binding protein 1, Chloride intracellular channel protein 1, Chromosome 14 open reading frame 166, yeast sds22 homolog and Prohibitin while those of eleven up-regulated were keratin type II cytoskeletal 4, Glyoxalase I, RuvB-link 2, HLA-B associated transcript (have two proteins) 1, Creatine kinase-B, HSP27, Proteasome activator subunit 3 isoform 1, PRDX6_HUMAN Peroxiredoxin 6, P58 and Cu/Zn-superoxide dismutase. Most of these de-regulated proteins are implicated in many types of cancers.
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