P116

Apoptosis by Cyanate in Osteosarcoma like Osteoblast

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Cyanate, which is derived spontaneously from urea at body temperature and pH, induces inflammation, fibrosis, and hemolysis. Cyanate, known as one of the uremic toxins, might also induces apoptosis. We studied the effect of cyanate on the apoptosis using osteoblastic cells. Osteoblastic ROS 17/2.8 cells, exposed to various concentrations of sodium cyanate, were used to analyze the apaototic factors such as caspases 3 and 8, PARP, FasL, c-IAP, and cytochrome c. Cyanate treatment caused the digestion of genomic DNA into ladders in concentration-dependent ways, associated with a decrease in intact DNA. The exposure to cyanate caused the expression of caspase proteins which associated with the activation of the degradation of 116-kDa PARP into a 85-kDa fragment in a concentration-dependent manner. Cyanate induced a marked decreased expression of pro-caspase 3 and pro-caspase8 protein. While, caspase 8 increased in intensity with a concentration-dependent manner. PARP, a substrate for caspase 3 and an important marker for apoptosis, was cleaved to the 85 kDa. Expression of FasL and c-IAP proteins was decreased. Cytochrome c release was released with dose-dependent. On the basis of these results, we suggest that cyanate is one of the contributing factors for apoptosis in ESRD patients.

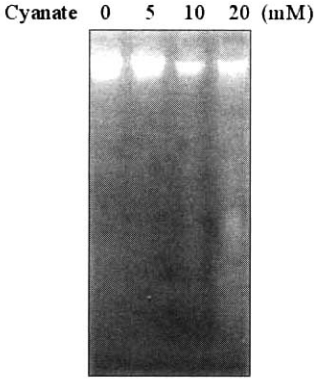

Fig. 1. *Analysis of DNA integrity in cyanate-treated ROS 17/2.8 cells by agarose electrophoresis. ROS 17/2.8 cells were treated with different concentrations (5, 10, and 20 mM) of cyanate for 24 h. DNA in cells was extracted and electrophoresed through a 2% agarose gel and visualize by staining with ethidium bromide.*

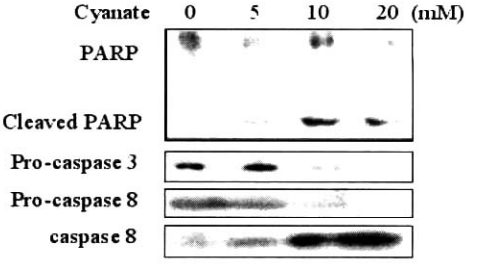

Fig. 2. *Effect of cyanate on pro-capase 3 and -8, and caspase 8, PARP expression in osteoblastic ROS 17/2.8 cells. Cells were treated with the indicated concentrations of cyanate for 24 h. The protein expression was determined by Western blot in cell lysates.*