

[PA4-14] [2003-10-10 09:00 - 13:00 / Grand Ballroom Pre-function]

Colchicine poisoning

Lee Juseon^o, Choi Hwakyung, Choi Hyeyoung, Woo Sanghee, Park Yoosin, Chung Heesun
National Institute of Scientific Investigation

Colchicine is a drug well known for its use in the therapy of acute attacks of gouty arthritis. Two death caused by oral ingestion of colchicine was reported. Two persons were all women. The one was 34 years old woman and with his boyfriend in a inn and the other was a housewife. The first one was a alcoholic but his boyfriend was afflicted with gout. So her boy friend carried over about 60 tablets of colchicine, 4 tablets of naltrexone and 22 tablets of fluoxetine. After her death there were remained colchicine 2 tabs, naltraxone 4 tabs and fluoxetine 22 tabs. We deduced that she ate her boy friend's colchicine about 58tabs with alcoholic drink. But when we met her blood, there was no alcohol but colchicine was detected in her blood. The blood concentration of colchicine was 0.2 μ g/ml. The second woman was not a alcoholic but she drank alcohol with her husband. After that she had a quarrel with her husband. She was placed under medical care for her ingestion of drug before her death. But in her blood there was no alcohol and colchicine. Colchicine in blood was extracted by liquid extraction. we analysed the colchicine with HPLC/PDA.

[PA4-16] [2003-10-10 09:00 - 13:00 / Grand Ballroom Pre-function]

Different mechanisms mediate uptake of lead in a rat glial cell line

Cheong Jae Hoon^o, Tan Blendyl, Kim Y.B., Bannon Desmond, Olivi Luisa, Bressler Joseph
Department of Pharmacy, Samyuk University and Kennedy-Krieger Institute, Johns Hopkins Univ., Baltimore, MD, 21205

The mechanism by which lead enters glial cells was examined. The uptake of lead reached saturation when assays were performed in buffers at pH 5.5 and 7.4. The Vmax and Km was 2.7 pmoles/mg protein/min and 13.4 M in the buffer at pH 7.4, respectively, whereas the Vmax and Km was 329 fmoles/mg and 8.2 M in the buffer at pH 5.5, respectively. Uptake in a buffer at pH 5.5 but not at pH 7.4 was inhibited by iron. Cells treated with the iron chelator desferoxamine displayed higher levels of the divalent metal transporter mRNA and protein. Cells treated with desferoxamine displayed greater uptake of lead in the buffer at pH 5.5 but in the buffer not at 7.4. The transport of Pb was blocked by the anion transporter inhibitor 4,4-diisothiocyanatodihydrostilbene-2,2-disulfonic acid (DIDS) in the buffer at pH 7.4, which bound to cell surface proteins at concentrations that were similar to those that blocked Pb uptake. DIDS did not inhibit uptake in the buffer at pH 5.5. Greater uptake of Pb was observed in a buffer containing sodium bicarbonate, which was inhibited by DIDS. When the uptake of Fe, Mn, and Zn was examined, only uptake of Zn was inhibited by DIDS. In summary, glial cells display two distinct transport mechanisms for Pb that are distinguishable by their sensitivity to inhibitors and activators at pH 5.5 and pH 7.4 in glial cells.

[PA4-17] [2003-10-10 09:00 - 13:00 / Grand Ballroom Pre-function]

New HDAC inhibitor, IN2001 induces apoptosis/cell cycle arrest in human breast cancer cells

Joung Ki Eun^o, Min Kyung Nan, Cho Min Jung, An Jin Young, Kim Dae Ki, Sheen Yun Yhong
Ewha Womans University, College of Pharmacy

The acetylation of histone is one of the mechanisms involved in the regulation of gene expression and is tightly controlled by two core enzymes, histone acetyltransferase (HAT) and deacetylase (HDAC). There are several reports that imbalance of HAT and HDAC activity is associated with abnormal behavior of the cells in morphology, cell cycle, differentiation, and carcinogenesis. Recently, an increasing number of structurally diverse HDAC inhibitors have been identified that inhibit proliferation and induce differentiation and/or apoptosis of tumor cells in vivo and in vitro. In this study, we have investigated the effects of novel HDAC inhibitors, IN2001 on ER positive and ER negative human breast cancer cell lines. The growth inhibition, cell cycle arrest and apoptosis of cells by HDAC inhibitors were determined using SRB assay, DNA fragmentation, and flow