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Objective and Design: The underlying mechanism of HCl in oesophagitis caused by the reflux of gastric juice, especially HCl, remains unclear. To investigate the underlying mechanism of HCl in oesophagitis, we observed the inflammatory responses to HCl in RBL-2H3 mast cells.
Materials and Methods: The rat basophilic leukemia (RBL-2H3) cells were used for measurements of histamine release, arachidonic acid (AA) release and reactive oxygen species (ROS) and peroxynitrite generation induced by HCl.
Results: Exogenous HCl dose-dependently increased the histamine release and ROS generation, whereas it decreased spontaneous release of [3H] AA and spontaneous production of peroxynitrite. Mepacrine (10 μM), oleyloxyethyl phosphorylcholine (10 μM) and bromoenol lactone (10 μM) did not affect both histamine release and ROS generation induced by HCl. U73122 (1 μM), a specific phospholipase C (PLC) inhibitor did not have any influence on histamine release and ROS generation. Propranolol (200 μM), a phospholipase D (PLD) inhibitor, and neomycin (1 mM), an nonspecific PLC and PLD inhibitor, significantly inhibited both histamine release and ROS generation. Diphenyleneiodonium (10 μM), an NADPH oxidase inhibitor, and tiron (5 mM), an intracellular ROS scavenger significantly inhibited HCl-induced histamine release and ROS generation.
Conclusion: These findings suggest that inflammatory responses to HCl is related to histamine release and ROS generation, and that ROS generation by HCl may be involved in histamine release via PLD pathway in RBL-2H3 cells.

Poster Presentations - Field B3. Neuroscience

[PB3-1] [10/18/2001 (Thr) 14:00 - 17:00 / Hall D]

Effects of tributyltin compounds on catecholamine biosynthesis in PC12 cells.

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The effects of tributyltin (TBT) compounds on dopamine biosynthesis in PC12 cells were investigated. Treatments of PC12 cells with tributyltin acetate (TBTA) and tributyltin chloride (TBTC) showed 42.8% and 44.9% inhibition of dopamine content at a concentration of 0.1 μM and 0.5 μM for 48h. IC50 values of TBTA and TBTC were 0.12 μM and 0.6 μM, respectively. Next, the intracellular mechanisms of TBT compounds were examined. Dopamine content decreased at 6h and reached a minimal level at 24h after the exposure to 0.1 μM TBTA and 0.5 μM TBTC. The decreased dopamine level was maintained for up to 48h. TH activity was inhibited at 6h following the treatment with TBT compounds and was maintained at a reduced level for up to 36h (20-40% inhibition at 0.1 μM of TBTA and 0.5 μM of TBTC). TH mRNA level also started to decrease at about 6h and reached a minimal level at 24h after exposure of PC12 cells to TBT compounds. These results suggest that TBT compounds contribute to the decrease in dopamine content by the inhibition of TH activity and the regulation of TH gene expression in PC12 cells.
* This work was supported by the Brain Korea 21 project.

[PB3-2] [10/18/2001 (Thr) 14:00 - 17:00 / Hall D]

Effects of tributyltin compounds on L-DOPA-induced neurotoxicity in PC12 cells.