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Background: It have been suggested that mast cells may play a role in the pathogenesis of ulcerative colitis (UC). To better define the role of mast cells in UC, we examined PAR2, tryptase, and TNF-α expression in normal tissue and UC tissue.

Methods: PAR2, tryptase, and TNF-α expression in 9 normal and 9 UC tissues were examined by immunohistochemistry.

Results: All of the three proteins were significantly more detectable in UC tissue than in normal tissue. Approximately 70.3 % of PAR2-positive lamina propria cells and 66.4 % of TNF-α-positive lamina propria cells were tryptase-positive mast cells, respectively.

Conclusions: These results show that PAR2-positive mast cells and TNF-α-positive mast cells may play an important role in the pathogenesis of UC.

[PB2-8] [10/18/2001 (Thr) 14:00 - 17:00 / Hall D]

Interrelation among Arachidonic Acid Release, Reactive Oxygen Species and Peroxynitrite Generation Induced by Silica in RAW 264.7 Cells

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Objective and Design: To investigate the underlying mechanism of silica in inflammatory response, we examined the interrelation among arachidonic acid (AA), reactive oxygen species (ROS) and nitric oxide (NO) in RAW 264.7 cells stimulated by silica.
Materials and Methods: RAW 264.7 cells were used for measurements of AA release, ROS, peroxynitrite (PON) generation and NO production elicited by silica. The effects of various inhibitors related to phospholipase (A2, C and D) pathway and ROS generation were observed.
Results: Silica dose-dependently increased [3H]AA release, ROS, PON generation and NO production. OPC (10 μM), DTT (5 mM) and MAFP (10 μM), significantly inhibited [3H]AA release, ROS and PON generation induced by silica. U73122 (a specific PLC inhibitor, 1 μM), neomycin (an nonspecific PLC and PLD inhibitor, 1 mM) and propranolol (a PLD inhibitor, 200 μM) significantly inhibited [3H]AA release and PON generation but did not inhibit ROS generation induced by silica. Diphenyleneiodonium chloride (10 μM), an NADPH oxidase inhibitor, and tiron (5 mM), an intracellular ROS scavenger, significantly inhibited [3H]AA release, ROS generation and PON generation induced by silica. NOS inhibitors, such as 1 mM L-NAME, 1 mM L-NNA and 1 mM L-NMMA significantly inhibited silica-induced PON production, but did not affect [3H]AA release and ROS generation induced by silica.
Conclusion: These results suggest that both AA release and ROS elicited by silica stimulate each other and these seem to be the upstream mediators in PON generation in RAW 264.7 cells.

[PB2-9] [10/18/2001 (Thr) 14:00 - 17:00 / Hall D]

Histamine Release by Hydrochloric Acid is Mediated via Reactive Oxygen Species Generation and Phospholipase D in RBL-2H3 Mast Cells

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