potent than that in C48/80-induced histamine release. EGTA dose-dependently inhibited ATP and C48/80-induced histamine release, but C48/80-induced histamine release was slightly inhibited by high concentrations (>2mM) of EGTA. Bisindolylmaleimide (protein kinase C antagonist) dose-dependently inhibited ATP and C48/80-induced histamine release. Calmodulin antagonists (W-7, trifluoperazine) had a little effect in ATP and C48/80-induced histamine release at low concentrations(<3μM), but at high concentration (W-7, >10μM; trifluoperazine, >3μM) they stimulated ATP and C48/80-induced histamine release. Tyrosine kinase inhibitors (methyl 2,5-dihydroxycinnamate, genistein) dose-dependently inhibited ATP and C48/80-induced histamine release. Protein kinases (such as protein kinase C, calmodulin-dependent pathway and tyrosine kinase) seem to be involved in histamine release induced by ATP and C48/80. These results suggest that ATP-induced histamine release is related to both intracellular calcium release and extracellular calcium influx via voltage-dependent calcium channel and receptor-operated calcium channel. C48/80-induced histamine release is related to extracellular calcium influx, especially by receptor-operated calcium channel rather than voltage-dependent calcium channel.

[PB1-3] [10/20/2000 (Fri) 15:30 - 16:30 / [Hall B]]

Effect of Four-Week Ethanol Intake on Exocrine Pancreatic Secretion in Rats

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To investigate the effect of long-term ethanol intake on pancreatic exocrine secretion, rats were freely accessed to 5% (w/v) ethanol instead of water for 4 weeks. These rats consumed approximately 2.5-3.0 g of ethanol daily. Pancreatic juice secretion rate was 25.4 2.3 l/hr in normal rats and 23.1 1.5 l/hr in ethanol-fed rats. Amylase activity and phospholipase A2 activity of pancreatic juice in normal rats were similar to those in ethanol-fed rats. However, protein concentration of pancreatic juice in ethanol-fed rats was significantly greater than that in normal rats. In acute pancreatitis induced by CBPD obstruction, water contents, amylase activity and phospholipase A2 activity of pancreatic fragment significantly increased. These increases indicate typical inflammatory response as acute pancreatitis. In ethanol-fed rats, water contents and amylase activity also significantly increased but phospholipase A2 activity did not as compared with that in normal rats. The obstruction of CBPD resulted in the increase of protein concentration, amylase activity and phospholipase A2 activity in serum. In ethanol-fed rats, serum protein concentration was significantly higher than that of normal rats, whereas amylase activity and phospholipase A2 activity were similar to those of normal rats. This result suggests that long-term intake of 5% ethanol like beer may cause the pancreatic inflammation.

[PB1-4] [10/20/2000 (Fri) 15:30 - 16:30 / [Hall B]]

The role of PKC activation on hypoxia-induced cell death in cardiomyocyte and smooth muscle cells

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Protein kinase C (PKC) has been known to play a role in the protective effect of ischemic preconditioning in heart and vascular cells. On the basis of the fact that high glucose can produce PKC activation, in the present study, we investigated whether high glucose (22 mM) can produce protective effect against hypoxia-induced cell death and whether the effect of high glucose is correlated with PKC activation in H9c2 cardiomyocyte and A7r5 smooth muscle cells.
The lactate dehydrogenase(LDH) release is estimated as a parameter of cellular injury. Apoptotic cell death was examined by the method of TUNEL(Terminal Deoxynucleotidyl-transferase Nick-End Labeling) staining.