

It was presented that one of the pancreatic enzymes, trypsin, modulates many biological processes by acting on specific proteinase-activated receptor 2 (PAR-2). PAR-2 belongs to a family of G protein coupled receptors activated by tethered ligand sequences within the N-terminal, which is made accessible after the site-specific cleavage of the protein. Trypsin activates PAR-2 by the mediation of a unique process inhering in the recognition of the receptor by enzyme, subsequent cleavage at the specific site of NH<sub>2</sub>-terminal and presentation of a new NH<sub>2</sub> terminal, which behaves as a tethered ligand. This ligand interacts with the extracellular domain of receptor molecule. Thus, PAR-2 is a receptor, whose ligand is a physical part of the receptor molecule. This receptor was previously described on normal as well as malignant immunocompetent cells, on endothelial and muscle cells of major as well as minor vessels. Its presence was also immunohistochemically demonstrated on intestinal epithelial cells, epithelial cells of exocrine organs, keratinocytes, fibroblasts and other cell types in stomach, small intestine, colon, liver and kidney. PAR-2 is expressed on various cells with a wide spectrum of cellular responses after activation. In the first part of this work we focused on the role of PAR-2 during the process of acute pancreatitis. An animal model of acute pancreatitis induced by taurocholate injection to ductus choledochus of Wistar rats was used. Much higher positivity for PAR-2 on acinary duct cells was observed in APL induced animals than in controls. Similar findings were noticed on arterial smooth muscle cells. Surprisingly, parallel to the exocrine pancreas and vessel findings, enhanced Langerhans' islets cell positivity was observed in experimental animals.