BMC Pharmacology

Meeting abstract

Differential anti-inflammatory properties of peroxisome proliferator-activated receptors (PPAR) α and γ in experimental pancreatitis

Thomas Griesbacher*, Veronika Pommer, Rufina Schuligoi and Bernhard A Peskar

Address: Institute of Experimental and Clinical Pharmacology, Medical University of Graz, Austria

Email: Thomas Griesbacher* - thomas.griesbacher@meduni-graz.at

* Corresponding author

from 13th Scientific Symposium of the Austrian Pharmacological Society (APHAR). Joint Meeting with the Austrian Society of Toxicology (ASTOX) and the Hungarian Society for Experimental and Clinical Pharmacology (MFT)
Vienna, Austria. 22–24 November 2007

Published: 14 November 2007

BMC Pharmacology 2007, **7**(Suppl 2):A20 doi:10.1186/1471-2210-7-S2-A20

This abstract is available from: http://www.biomedcentral.com/1471-2210/7/S2/A20

Perfluorooctanoic acid (PFOA) has anti-inflammatory effects in models of cutaneous inflammation, possibly via activation of PPAR-α and PPAR-γ. We have therefore investigated whether PFOA has similar effects in a model of acute oedematous pancreatitis and whether such effects could be explained by agonism at PPAR-α or PPAR-γ. Acute pancreatitis was induced in anesthetized rats by i.v. infusion of the cholecystokinin analogue, caerulein. The PPAR-α agonist clofibrate or the PPAR-γ agonist rosiglitazone were injected s.c. before the experiments. Pancreatic oedema, neutrophil activation and production of prostaglandin (PG) E_2 and prostacyclin (via 6-keto-$PGF_{1\alpha}$) were assessed in the pancreas. Acute pancreatitis caused significant oedema formation, neutrophil activation as assessed by myeloperoxidase activity in the tissue, and increased synthesis of pro-inflammatory prostanoids. Neutrophil activation was unaffected by clofibrate but was abolished by rosiglitazone. In contrast, prostanoid synthesis was unaffected by rosiglitazone but was inhibited by clofibrate. In conclusion, our data demonstrate that activation of PPAR-α and PPAR-γ has differential anti-inflammatory effects in acute interstitial-oedematous pancreatitis. Neutrophil activation is sensitive to inhibition by PPAR-γ activation while the production of pro-inflammatory prostanoids can be attenuated by activation of PPAR-α. Thus, the anti-inflammatory potential of PPAR-α and PPAR-γ ligands should be further investigated.