BMC Pharmacology

Meeting abstract

The role of cannabinoid CB_1 receptor agonists in gastric mucosal protection in rats and mice

Nashwan Shujaa[1], Zoltán S Zádori[1], Mahmoud Al-Khrasani[1], Susanna Fürst[1], Tibor Wenger[2] and Klára Gyires*[1]

Address: [1]Department of Pharmacology and Pharmacotherapy, Semmelweis University, Budapest, Hungary and [2]Department of Human Morphology, Semmelweis University, Budapest, Hungary

Email: Klára Gyires* - gyirkla@pharma.sote.hu

* 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):A47 doi:10.1186/1471-2210-7-S2-A47

This abstract is available from: http://www.biomedcentral.com/1471-2210/7/S2/A47

CB_1 receptor agonists inhibit stimulated gastric acid secretion and exert an anti-ulcer activity in acid-dependent ulcer models. The aims of this study were to investigate the gastroprotective effect of cannabinoids in an acid-independent ulcer model and to analyze the role of opioid and vanilloid receptors in this effect. Gastric mucosal damage was induced by acidified ethanol in rats and in $CB_1^{+/+}$ and $CB_1^{-/-}$ mice. Anandamide, methanandamide and WIN-55,212-2 inhibited the ethanol-induced gastric mucosal damage significantly after peripheral and central administration, and their effects were reversed by the CB_1 receptor antagonist SR141716A. The gastroprotective effect of cannabinoid agonists was significantly decreased by naloxone and partially by capsazepine (TPRV1 receptor antagonist). The gastroprotective effect of opioid peptides DAGO and deltorphin II was significantly reduced in $CB_1^{-/-}$ mice. In conclusion, cannabinoid CB_1 receptors are likely to be involved in gastric mucosal defense. The effect seems to be central, and correlation between opioid and cannabinoid system in gastric mucosal protection may be raised.

Acknowledgements

This work was supported by ETT 529/2006.