BMC Pharmacology



Meeting abstract Open Access

Potential role of nociceptin and nocistatin in central regulation of gastric mucosal defense

Zoltán S Zádori¹, Nashwan Shujaa¹, Kornélia Tekes² and Klára Gyires*¹

Address: ¹Department of Pharmacology and Pharmacotherapy, Faculty of Medicine, Semmelweis University, Budapest, Hungary and ²Department of Pharmacodynamics, Faculty of Pharmacy, 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):A44 doi:10.1186/1471-2210-7-S2-A44

This abstract is available from: http://www.biomedcentral.com/1471-2210/7/S2/A44

© 2007 Zádori et al; licensee BioMed Central Ltd.

Nociceptin (NOC) and Nocistatin (NS) are two neuropeptides derived from pre-pro-nociceptin. NOC is the endogenous ligand of the NOP receptor which exhibits marked structural analogy with opioid receptors. NOC has been reported to exert multiple effects in the gastrointestinal tract. The aims of this study were to compare the gastroprotective effects of NOC and NS and to analyse the mechanism of their gastroprotective action. Gastric mucosal damage was induced by acidified ethanol in rats. The compounds were given i.c.v. Both NOC and NS (0.2– 5 nmol/rat) induced gastroprotective effects. Pre-administration of NS significantly decreased the effect of NOC, as well as the competitive antagonist J-113397 (70 nmol/ rat) and vagotomy. The effect of both neuropeptides was reduced by naloxone (27 nmol/rat), naltrindole (4.8 nmol/rat), norbinaltorphimine (10 μg/rat) and β-FNA (20 nmol/rat). In conclusion, both NOC and NS initiate centrally a series of events which result in gastric mucosal defense. The gastroprotective effect of NOC and NS is likely to be mediated by endogenous opioids and conveyed to the periphery by a vagal-dependent mechanism.

Acknowledgements

This work was supported by ETT 529/2006.