Role of substance P and CGRP in PAR-2-induced rectal hyperalgesia in rats

Bruno Guiard, Nicolas Cenac, Rafael Garcia Villar, Nathalie Vergnolle, Anne-Marie Coelho, Jean Fioramonti, Lionel Bueno

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Bruno Guiard, Nicolas Cenac, Rafael Garcia Villar, Nathalie Vergnolle, Anne-Marie Coelho, et al.. Role of substance P and CGRP in PAR-2-induced rectal hyperalgesia in rats. 2001 A Brain-Gut Odyssey, Jul 2001, Oxford, Angleterre, United Kingdom. Digestion, 2001, Digestion. 10.1159/000051897 . hal-01604751

HAL Id: hal-01604751

<https://hal.science/hal-01604751>

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Digestion 2001;63:247–279

<https://doi.org/10.1159/000051897>

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Digestion 2001;63:247–281

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H. Raybould, Davis, Calif., USA
F. Azpiroz, Barcelona, Spain

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Article / Publication Details

Published online: June 29, 2001
Issue release date: 2001

Number of Print Pages: 33

Number of Figures: 0

Number of Tables: 0

ISSN: 0012-2823 (Print)

eISSN: 1421-9867 (Online)

For additional information:

<http://www.karger.com/DIG>

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"2001 A BRAIN-GUT ODYSSEY" | In: DIGESTION 63, p.275.
Oxford, UK, July 11-15, 2001.

ROLE OF SUBSTANCE P AND CGRP IN PAR-2-INDUCED RECTAL HYPERALGESIA IN RATS

Intracolonic infusion to rats of the proteinase-activated receptor-2 (PAR-2) activating-peptide SLIGRL, induced delayed hyperalgesia to rectal distension (RD). This study investigates the involvement of substance-P (SP) and calcitonin gene-related peptide (CGRP) in PAR-2-induced visceral hyperalgesia.

Methods: Four groups (n=6-8) of male Wistar rats (250-300g) were equipped with an intracolonic (IC) catheter for SLIGRL (0.2mg/rat) or saline (0.25ml/rat) infusions, and electrodes for recording abdominal contraction (mean±SEM for 5-min episodes) as visceral pain index. RD was performed by 5min steps of 0.4ml (0 to 1.2mL). Groups 1&2 were infused with SLIGRL or saline (0.25mL/20min) respectively, and the NK1-antagonist SR140333 (1mg/kg-IP): first, SR140333 was administered 30min before SLIGRL or saline; second, 3 days later, SR140333 was administered 10h after SLIGRL or saline. Groups 3&4 were similarly infused with SLIGRL or saline respectively, and the CGRP receptor-antagonist human-CGRP₈₋₃₇ (100 µg/kg-IV), 20min before and 10h after SLIGRL or saline.

Results: SLIGRL (group 1) but not saline (group 2) induced 10h-delayed visceral hyperalgesia to RD (45.0±4.1 contractions vs 33.3±3.0 in basal conditions i.e. 36% increase; p<0.05). SR140333 inhibited SLIGRL-induced hyperalgesia when administered before (45.0±4.1 vs 31.2±4.5; p<0.05) but not after SLIGRL (45.0±4.1 vs 48.0±4.8; p>0.05).

Similarly, SLIGRL (group 3) but not saline (group 4) increased the number of abdominal contractions by 47% in response to RD (1.2mL) at 10h post-infusion. hCGRP₈₋₃₇ did not affect SLIGRL-induced hyperalgesia when administered either before or after SLIGRL.

Conclusion: NK1-receptor blockade prevents SLIGRL-induced visceral hyperalgesia which suggests that SP, but not CGRP, is involved in PAR-2-induced sensitization of primary afferent neurons.

B. Guiard, N. Cenac, R. Garcia-Villar, A.M. Coelho, J. Fioramonti, L. Bueno

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