

Normal basal motility and enhanced cholinergic reactivity of the jejunum in nematode-induced hypermastocytosis in rats

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[Resume : 63112] Poster # 1572 NORMAL BASAL MOTILITY ENHANCED CHOLINERGIC AND REACTIVITY OF THE **JEJUNUM** NEMATODE-INDUCED IN HYPERMASTOCYTOSIS IN RATS

J. Gay, J. Fioramonti, R. Garcia-Villar, L. Buéno. Dept Pharmacology, INRA,

An increase in intestinal mast cell number (hypermastocytosis) has been observed in several digestive diseases including inflammatory bowel diseases and irritable bowel syndrome. Both the anatomical proximity of nerves to mast cells, and the functional mast cell-nerve communication occurring in both directions, have lead us to investigate whether intestinal motility is modified in intestinal hypermastocytosis. The aim of this study was therefore, to compare basal and carbachol-stimulated intestinal motility in control rats and in rats previously infected with Nippostrongylus brasiliensis (Nb) which are characterized by a jejunal hypermastocytosis persisting several weeks after the worm expulsion, occuring on the 3rd post-infection week.

Methods: 12 male Wistar rats were infected subcutaneously (s.c.) with 5000 third-stage larvae of Nb. 12 control rats received a s.c. injection of sterile NaCl 0.5%. 30 days later, rats were equipped with 3 groups of nichrome electrodes implanted 10 cm apart in mid jejunum. Recordings of jejunal myoelectric activity with an electroencephalograph were started 5 days after surgery. 10 days later, rats were sacrificed and samples of jejunum taken for histological staining with hemalun and eosin for routine examination and with Alcian Blue-safranin for identification of mast cells. The motility parameters analyzed were the frequency and velocity of propagation of migrating myoelectric complexes (MMC), and the duration of their disruption after a standard meal (3 g) or 0.1 mg/kg, s.c., carbachol administration. Values were means \pm SEM. They were compared using ANOVA and Student's "t" test for

Results: Inflammatory sites were not detected, macroscopically microscopically, in intestinal samples from both control and post-infected rats. Jejunal mast cell numbers were $69 \pm 26 \text{ /mm}^2$ in controls and reached 303 ± 97 /mm² in post-infected rats. In control rats, MMC frequency and velocity of propagation were 3.9 ± 1.0 /h and 1.24 ± 0.09 cm/min, respectively; postprandial MMC disruption after a 3 g meal lasted 143 ± 27 min. These values were not significantly different (p>0.05) in post-infected rats $(4.0 \pm 1.1 \text{ MMC/h}, 1.30 \pm 0.07 \text{ cm/min}, 185 \pm 32 \text{ min})$. Carbachol (0.1 mg/kg s.c.) disrupted the MMC pattern which was replaced by an intense irregular activity for 72 ± 5 min, a value which attained 125 ± 8 min in postinfected rats (p<0.0001 vs controls).

Conclusions: These results indicate that, in the absence of inflammation, intestinal hypermastocytosis does not modify basal fasted and postprandial patterns of intestinal motility, but strongly enhances the duration of the contractile response to a cholinergic stimulation.

I affirm that this abstract meets all of the requirements stated in this booklet, that the authors named are familiar with the presented data, and that box B has been signed. On behalf of all of the authors, I hereby transfer copyright in the above abstract to American Gastroenterological Association.

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