Format for Scientific Research Process

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Title: Corticotropin-releasing factor stimulates colonic motility *via* muscarinic receptors in the rat

Authors: Kyung-Jo Kim, Ki Bae Kim, Soon Man Yoon, Joung-Ho Han, Hee Bok Chae, Seon Mee Park, Sei Jin Youn

Correspondence to: **Sei Jin Youn, MD,** Department of Internal Medicine, Chungbuk National University College of Medicine, 410 Seong bong-ro, Heungduk-gu, Cheongju, Chungcheongbuk-do 361-711, South Korea. sjyoun@chungbuk.ac.kr

Telephone: +82-43-2696357

Fax: +82-43-2733252

1 What did this study explore?

This study was conducted to explore exogenous corticotropin-releasing factor (CRF)-induced motility in the isolated rat colon and to demonstrate the effect of pharmacologic inhibition on CRF-induced motility in the same model.

2 How did the authors perform all experiments?

First, we developed the isolated rat colon model, which is free from control by the autonomic nervous system, and the enteric nervous system and regulatory systems (endocrine, paracrine) remain functional. Thus, the model may be optimal for investigating colonic motility. Second, corticotrophin releasing factor was administered

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in a stepwise manner. Third, each medication, such as phentolamine, propranolol, hexamethonium, tetrodotoxin, and atropine, was infused, after recording baseline colonic motilities. Then, corticotropin-releasing factor was given simultaneously in the isolated colon.

3 How did the authors process all experimental data?

The primary outcome of this study was the motility index (MI). Data are presented as means ± SE (standard errors). Kendall's rank correlation coefficient was used to measure the association between drug concentration and motility response. Because of the small sample size, continuous variables, such as 'CRF' and 'CRF and atropine', were compared by the Mann-Whitney *U* test. A *P*-value of < 0.05 was considered statistically significant.

4 How did the authors deal with the pre-study hypothesis?

Although the role of CRF in colonic motility is well-established, the data on pharmacological inhibition in CRF-induced colonic motility is scant.

5 What are the novel findings of this study?

CRF-induced colonic motility appears to be mediated by local cholinergic signaling via muscarinic receptors. Muscarinic receptors are potential targets for counteracting CRF-induced colonic hypermotility.

Sincerely,
Kyung-Jo Kim, MD, Ph D.