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Abnormal colonic cholinergic and nitrergic activities in relation to elastosis in uncomplicated diverticular disease

Mark Golder

Mark Golder, Center for Academic Surgery, Institute of Cell and Molecular Science, Barts and the London School of Medicine and Dentistry, Queen Mary University, London E11BB, United Kingdom
Author contributions: Golder M wrote this paper.
Correspondence to: Mark Golder, PhD, FRCS (Eng), FRCS (Gen), Center for Academic Surgery, Institute of Cell and Molecular Science, Barts and the London School of Medicine and Dentistry, Queen Mary University, London E11BB, United Kingdom. email@msgolder.co.uk
Telephone: +44-20-73777079 Fax: +44-20-73777283
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Abstract

I read with interest the review on the pathogenesis of diverticular disease by Commane *et al* in *World J Gastroenterol* 2009; 15(20): 2479-2488. However, I would like to discuss several important errors that the authors made whilst citing information from previously published work on the neuromuscular dysfunction in the disease.

Key words: Diverticular disease; Neural; Acetylcholine; Nitric oxide; Elastin; Protein gene product

Peer reviewers: Emiko Mizoguchi, MD, PhD, Department of Medicine, Gastrointestinal Unit, GRJ 702, Massachusetts General Hospital, Boston, MA 02114, United States; Frank Hoentjen, MD, PhD, Department of Gastroenterology, VU Medical Center, Sumatrastraat 16, 2022XL Haarlem, The Netherlands

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TO THE EDITOR

I read with interest the review on the pathogenesis of diverticular disease by Commane *et al*[1] in *World J Gastroenterol* 2009 May 28; 15(20): 2479-2488. However, in the discussion, the authors made several important errors while citing information from previously published work on the neuromuscular dysfunction in the disease. The authors argued that methods for measuring the general nerve, and cholinergic and nitrergic activity in longitudinal muscle (LM) in the disease[2,3] may have been erroneous because of potential confounders of elastosis and smooth muscle shortening, and mentioned that it was not clear how these potential confounders were controlled.

First, the authors stated in error that these studies used prostaglandin as a marker of general nerve tissue, whereas in fact, both studies reported the antibody localization of protein gene product (PGP), a marker of general nerve tissue, which has been used extensively to localize nerves in histological sections[4].

As discussed previously[3], the reduction in immunoreactivity of PGP in LM in diverticular disease was probably a spurious finding, secondary to the associated 200% increase in the surface area of elastin in LM compared with normal controls. It may be not due to an increase in general nerve degeneration in the disease, as the qualitative signs of degeneration were similar between the disease and controls, and reflected the mean age of the patients.

The method that was used to semi-quantify cholinergic and nitrergic activity was specifically designed to overcome the potential problem of the effects of elastosis of LM in the disease. Choline-acetyl-transferase (ChAT), a marker of cholinergic activity, and nitric oxide synthase 1 (NOS1), a marker of nitrergic activity, were each co-localized with PGP on histological sections, which then underwent immuno-fluorescence analysis. The immuno-reactivities were expressed as % (surface area of ChAT)/% (surface area PGP) and (surface area of NOS1)/% (surface area PGP), respectively. The finding of lower immuno-reactivities of

both ChAT and NOS1 in diverticular LM, compared with controls, was therefore considered non-spurious, as any potential effect of elastosis in diluting immuno-reactivity would have been the same for both the neurotransmitters and PGP. These arguments were discussed in the respective articles[2,3].

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