

## Letter: is *Helicobacter pylori* behind Barrett's oesophagus and colorectal neoplasms?

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SIRS, Andrichi *et al.*'s meta-analysis<sup>1</sup> showed that Barrett's oesophagus (BO) was associated with an increased risk of both colorectal adenomas and colorectal cancer (CRC). They concluded that if the risk estimates for CRC in BO patients reflects a real relationship, an established association will warrant a search for common genetic or environmental risk factors.

Some genetic alterations are common in both conditions<sup>2</sup>; we initially found that specialised intestinal metaplasia indicating BO appeared in a significant percentage of patients with colon tumours (12/23) compared with controls (2/14) and was associated with increased oesophageal mucosal expression of oncogenes Ki-67 and p53/Bcl-2<sup>3</sup> that indicated mainly increased proliferation leading to oncogenesis.

In this regard, *Helicobacter pylori* infection may be considered a promoter of both diseases. Our and others' data<sup>4</sup> indicate that *H. pylori* infection might contribute to oesophageal adenocarcinoma progress in subpopulations with gastro-oesophageal reflux disease and BO. In this respect, gastrin, induced by *H. pylori* infection, is an oncogenic growth factor contributing to oesophageal, gastric and colon carcinogenesis and, in particular, playing a potential causal effect on neoplastic progression in BO and left side CRC showing, for instance, anti-apoptotic activity through upregulation of the anti-apoptotic Bcl-2 and stimulation of mutagenic and tumourigenic cyclooxygenase-2 expression.<sup>4-6</sup>

Moreover, *H. pylori* infection is mostly frequent in colonic adenomas and tumour tissues (documented by immunohistochemical stain) and is accompanied by increased cell proliferation (mainly enhanced Ki-67 and

Bcl-2 expression) and impaired apoptotic (decreased Bax) processes, thereby indicating its potential pathogenetic role.<sup>6-8</sup>

Apart from upper gastrointestinal tract (UGT), *H. pylori* infection might also cause chronic inflammatory colon mucosal damage and stimulate cancer stem cells and/or recruit bone-marrow-derived stem cells, which may ultimately facilitate UGT and colon tumour formation and progression.<sup>8-10</sup> However, further studies are needed to elucidate the proposed pathophysiological mechanisms involved in *H. pylori*-associated colon oncogenesis; its eradication might inhibit these oncogenic processes.

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