Letters to the Editor


Letter

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The conclusions drawn by the authors are misleading due to a number of reasons:

(a) It is not clear what gastrin species were measured in their assay system. Many authors have shown that when confounding factors such as Helicobacter pylori infection and use of proton pump inhibitors are taken into account, there is no rise in amidated gastrin levels [Penman et al. (1) and Ciccotosto et al. (2)]. However immature forms of gastrin, such as progastrin, are increased in colorectal cancer patients and have been shown to decrease following surgical resection of the tumor [Ciccotosto et al. (2) and Siddheshwar et al. (3)].

(b) Pancreatic cancer has been shown by a number of groups to secrete immature gastrin forms and have a potential autocrine/paracrine pathway [Caplin et al. (4), Smith et al. (5), Goetze et al. (6)], and it is therefore likely that such gastrin peptides are elevated in the serum.

(c) Interestingly, expression of both cholecystokinin and gastrin gene was shown both to be up-regulated in a gastric carcinoma cell line, with the gastrin gene being selectively transcribed [Goetze et al. (6) and van Solinge et al. (7)]. This suggests that cholecystokinin protein secretion by malignant cells is unlikely to contribute to serum levels.

References


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