

Letters to the Editor

Correspondence re: Weinberg *et al.*, Cholecystokinin and Gastrin Levels Are Not Elevated in Pancreatic Carcinoma. *Cancer Epidemiol. Biomark. Prev.*, 10: 721–722, 2001

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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 auto-crine/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.

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