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Short Communication

Inappropriate Removal of Gastric Acidity is due to Lack of Biological Respect

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During evolution, species develop functions to protect them from dangers like infections. Intact skin is impermeable towards most microorganisms and is accordingly central in this defense. In the gastrointestinal tract, however, there are opposite interests between absorption of nutrients and defense against infectious agents. Evolution has come to a compromise in covering the mucosa of the mouth and esophagus with squamous cell epithelium and producing acid in the first part of the gastrointestinal tract, the stomach, with a single layered epithelium. The gut epithelium is single layered and particularly thin in the terminal ileum with M-cells (specialized cells to allow uptake of antigens to present them for the immune apparatus).

The gastric juice is unique combining a high acidity with an active proteolytic enzyme, pepsin. Pepsin rapidly loses its proteolytic activity when pH exceeds 4.0 [1], which is also the minimal acidity normally seen even during meals [2] and the pH above which the bactericide activity of the gastric juice is lost [3].

Normal gastric juice kills not only bacteria, but also virus and probably also prions [4]. Taking into consideration the risk connected to the highly acidic gastric juice (peptic ulcer disease and reflux esophagitis), gastric acidity must obviously have been of importance during evolution. Preservation of acidic juice in the upper gastrointestinal tract during evolution from primitive fishes to humans also underlines the importance of this function.

Moreover, the hormone gastrin and the vagal nerves tightly regulate gastric acidity. Neural regulation (the cephalic phase) primes the stomach by stimulating acid secretion even before swallowing food, and therefore this mechanism is particularly developed in species tolerating contaminated food. In man, the gastric phase (gastrin) is quantitatively most important in stimulation of gastric acid secretion. Gastrin release reaches maximum at a pH around 4.0 [5] demonstrating the importance of sufficient gastric acidity. As is often the case, stimulation of function and growthis parallel; so also for gastrin in regulating the function and growth is (trophic effect) of its target cell, the ECL cell [6]. Continuous overstimulation of the ECL cell by hypergastrinemia will after decades predispose to gastric ECL cell derived neoplasia of variable malignancy [7,8].

Acidic gastric juice is a bactericide. Only Helicobacter pylori has been able to create a niche for itself by burying into the mucous layer, there produce NH3 by its urease activity, and thus establish a microenvironment where it can live. It has been claimed that in modern societies with a high hygienic standard, the need of gastric acidity is reduced, and that the evolution has not caught up with change in environment. However, we do not know the etiology of most of the chronic inflammatory diseases like ulcerative colitis, Crohn´s disease and rheumatoid arthritis. Moreover, even the neurodegenerative diseases like Alzheimer´s disease and Parkinson´s disease could be infectious caused by for instance prions [4].

The reason for the presumption that gastric acidity is no longer important is the experience with patients with hypo/anacidity due to atrophy of the oxyntic mucosa secondary to gastritis. However, these patients have an increased risk of gastric carcinoids (now called neuroendocrine tumors (NETs)) as well as carcinomas [7,8]. Furthermore, persons with reduced gastric acidity are susceptible to gastrointestinal infections [9,10]. Furthermore, atrophic gastritis whether due to so-called autoimmune gastritis or Helicobacter pylori gastritis, is primarily a disease of middle-aged or old people, and many infections and particularly neoplasia often have latency of decades.

Consequently, we should treat our patients with acid related diseases as carefully as possible.Firstly, patients should be properly diagnosed, and only those having an established diagnosis of ulcer disease or reflux esophagitis should be given inhibitors of gastric acid secretion. Since treatment with proton pump inhibitors induces rebound acid secretion [11] which may itself induce dyspeptic symptoms [12], it seems wise to start with the less efficient histamine-2 blockers in most cases, except those with severe disease. In older people, the risk of neoplasia with iatrogenic hypergastrinemia may probably be neglected.

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