Discussion

Card and Marks (2) found that generally acid output was proportional to the parietal cell numbers, with duodenal ulcer patients having high numbers of parietal cells. However, they found "that the parietal cell output differs so sharply in the duodenal ulcer and gastric disease groups, that the possibility must be entertained that the parietal cells in each of these groups may arise from two different populations".

We suggest that the differences in acid secretion in these groups are due to colonization of different areas of the stomach by *C.pylori*. The greatest reduction in acid output occurring when *C.pylori* colonizes the predominant acid secreting area - the body mucosa, an area where urease induced H^+ back-diffusion would have the greatest impact.

Heterotopic gastric mucosa may be present in the duodenum, and fundic glands can be present in the antrum. *C.pylori* colonizes such tissue. In prepyloric/antral and duodenal ulceration, urease induced back diffusion of H^+ in areas containing fundic glands may contribute to ulceration.

Conclusion

Differences in acid secretion in the various categories of peptic ulcer patients can be explained by differences in *C.pylori* colonization. This colonization could predispose to both gastric and duodenal ulceration via urease induced H^+ back diffusion dependent on the degree of heterotopia.

References

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Abstract No. 221

Protection of Campylobacter pyloridis (CP) but not Campylobacter jejuni (CJ) against acid susceptibility by urea

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CP colonization of the stomach is present in 30-60% of dyspeptic patients who undergo upper gastrointestinal endoscopy. CP is usually isolated by culture of a gastric biopsy, and in 20% of positive cases live organisms can be recovered from the gastric juice. However, the ability of the bacterium to survive in the range of acidity present in the stomach and the role

of its urease in its survival is uncertain. Therefore, we examined the pH susceptibility of CP for comparison with CJ in the presence and absence of 5 mM urea.

A heavy 2-3 day growth of CP was harvested from a horse blood agar plate (BAP) into 3 ml normal saline at pH 7.0 to give a cloudy suspension. 1:10 dilutions were made in phosphate buffered saline at pH's 1.5 to 7.0 and, after 30 minutes at 37°C, quantitative cultures were done on BAPs which were incubated for 3-5 days at 37°C in a microaerophilic environment. Sheep BAPs at 42°C were used for CJ cultures. We found that while CP survived well to pH 4-4.5, it was reduced by 3-5 logs at $\leq$pH 3.5.

CJ was similar in its acid susceptibility to CP. In striking contrast, the addition of 5 mM urea completely protected CP but not CJ from acidic pH's down to 1.5. This occurred in two ways: (1) by neutralizing the solution pH from $\geq$3.0 to $\geq$6.6 and (2) in addition, however, urea did not alter the highly acidic solution pH's of 1.5, 2.0 or 2.5 but still fully protected the CP organism. These findings show that urea allows the organism to protect its "microenvironment" in highly acidic solutions as well as provide a modest buffer in mildly acidic solutions. The high urease activity of CP provides it with the ability to survive in acid and thus may be an important prerequisite for colonization of the mammalian stomach.

Abstract No. 97

Identification of cytotoxic activity produced by Campybacter pylori

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Introduction

The mechanism by which *C.pylori* may contribute to development of upper GI tract disease is unknown. We sought to determine if *C.pylori* can produce a factor capable of altering cultured cells. We found broth culture filtrates of *C.pylor* induced intracellular vacuolization in cultured mammalian cells *in vitro.*

Materials and Methods

Broth culture filtrates of *C.pylori* were prepared by filtration (0.45 u) of culture supernatnat. Cultured cells were exposed to broth culture filtrates and cytotoxic effects were observed after 48 h by phase microscopy.