Campylobacter pylori, acid, and bile

Gastric acid rapidly destroys most bacteria, few surviving after 30 minutes exposure to a pH of less than 4·0,[1] and *Campylobacter pylori* is no exception.[2] Human gastric epithelium, however, is colonised by *C pylori* and ferret stomachs by a similar campylobacter-like organism, both organisms having a pronounced urease activity.[3] *C pylori* is uncommon in patients with histological evidence of reflux gastritis and increased gastric bile acid concentrations, but present in most patients with non-specific (type B) chronic gastritis.[4] Inhibition of growth of *C pylori* by 1% bile salts has been reported.[5] We studied the tolerance of gastric campylobacters to acid and bile.

Bacteria were suspended in physiological saline to give about 10^9 organisms ml^{-1}, and 50 μl of suspension was inoculated into tubes containing 1 ml of 0·1M citrate-phosphate buffer at various pH values (2·6–7·0). Paired tubes were inoculated, with and without 6 $mmol/l^{-1}$ urea. After 30 minutes of incubation at room temperature the suspensions were subcultured on to blood agar plates using a 1 μl loop. Plates were incubated microaerophilically at 37°C for 72 hours. There was confluent growth of *C pylori* (NCTC 11637 and two clinical isolates), two ferret gastric campylobacter-like organisms, *Escherichia coli* (NCTC 10418), and *Campylobacter jejuni* (NCTC 11392) from buffers at pH 7·0 to 4·0, and decreased survival below pH 4·0 with no growth of any bacteria at or below pH 3·0. Addition of urea had no effect on the survival of *E coli* or *C jejuni*. The three strains of *C pylori* were completely inhibited by a pH of $< 3{\cdot}6$, and two ferret gastric campylobacter-like organisms were inhibited by a pH of $< 3{\cdot}2$, but all five urease positive organisms survived exposure for 30 minutes at a pH of 2·6 when urea was added ($> 10^5$ organisms ml^{-1}), although no pH change was detected in the buffer. Non-parietal gastric juice contains urea at 5·3 $mmol/l^{-1}$.[6]

To test tolerance to bile, bacteria were cultured on blood agar plates (Oxoid blood agar base No 2 with 5% defibrinated horse blood) containing beef bile (Bacto oxgall, Difco) for 72 hours in appropriate atmospheric conditions. Clinical isolates of *Streptococcus faecalis* and *Clostridium perfringens* (*E coli* (NCTC 10418), *C jejuni* (NCTC 1168 and 11392) and *E coli* (NCTC 11366 and 11353) all grew well in the presence of 10% bile (10 g/l^{-1}, oxgall). Growth of *C pylori* (NCTC 11637), six clinical isolates of *C pylori*, and six strains of ferret gastric campylobacter-like organism were not affected by 0·1% bile, reduced by 0·5–1% and inhibited by 5% bile.

These experiments confirm the suggestion that urease activity enables gastric campylobacters to survive at low pH, when physiological amounts of urea are present, and that growth of these organisms is inhibited by concentrations of bile which are tolerated by other bacteria found lower in the gastrointestinal tract.[7] Intolerance of bile may be the reason for the absence of *C pylori* from the stomachs of patients with persistent biliary reflux and from the lower gastrointestinal tract.

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Cost effectiveness of postmortem histology

WA Reid suggests that unselected postmortem histology is not cost effective.[1] This may be so in terms of the likelihood of changing the main cause of death, but there are other valid reasons for performing postmortem histology which cannot be accommodated in such a simple cost benefit analysis.

Firstly, postmortem histology is a form of quality control over a pathologist's macroscopic diagnoses. One may not often find a mistake, but if routine histology is omitted it is easy for a busy pathologist to start believing that he or she is infallible.

Secondly, there are a host of minor abnormalities which are only detectable on histological examination and do not affect the main cause of death, but which are nevertheless important in building up the complete picture. In the spleen, for example, one thinks of the scattered granulomata of subclinical sarcoidosis, foci of extramedullary erythropoiesis, atheroma emboli, etc. In the adrenal it could be lesions in the capsular arteries, or "active" periadrenal brown fat. The only way a pathologist can recognise this type of lesion and assess its importance is by sampling a wide range of tissues routinely.

The idea that postmortem histology consists of "examining numerous rather uninteresting slides" is certainly not shared by me. We may well be entering an era of clinical budgeting, with corresponding pressure on pathology budgets including the postmortem budget. Pathologists should make it clear that a postmortem examination is much more than just the dissection of a body, and that the funding has to cover not only the postmortem room itself, but also a large slice of the histology budget—not to mention the budgets in microbiology, toxicology, and others.

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Dr Reid comments:

Dr Simpson appears to accept the main thesis of my paper that *unselected* postmortem histology seldom leads to a change of the final diagnosis. Both arguments he advances for taking it relate to its educational benefits, mainly to pathologists. Quality control is important, but a properly instituted system on random cases would be better than routine histology on every case. I accept that in the absence of routine histology incidental findings such as those he mentions would be missed; the question is whether their discovery justifies the effort of showing them. This is up to individual pathologists to decide, but, bearing in mind the considerable workload this imposes on medical laboratory scientific officers in preparing sections, I do not accept that this use of resources is worthwhile. The value of postmortem histology in the specific circumstances listed in the paper is not, of course, being questioned.

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