CORRESPONDENCE

Coccyoid Forms of *Helicobacter pylori*

To the Editor—Eaton et al. [1] recently described experiments with coccyoid *Helicobacter pylori* in gnotobiotic piglets. This interesting study examined the colonization and the reversion from coccyoid to bacillary *H. pylori* within 2 weeks of inoculation. Piglets colonized with bacillary *H. pylori* had more severe gastritis 2 weeks after inoculation than did those sacrificed 1 week after inoculation. The coccyoid *H. pylori* did not colonize stomachs and did not lead to histopathologic changes.

Our experience with coccyoid *H. pylori* inoculated intragastrically in a similar experimental design in BALB/c mice differed [2]. Concentrated coccyoid (*A_{550} = 5\) McFarland units) *H. pylori* cultures (20 days of incubation), not culturable in vitro (0 cfu/mL), showed colonization and produced gastric alteration with a peak 4 weeks after inoculation. In our studies, the histologic changes obtained with fresh coccyoid *H. pylori* were lower than those in mice infected with the same strains in bacillary morphology, probably due to partial reversion of the coccyoid bacteria and therefore smaller concentrations of strains colonizing the mouse stomach.

Others have shown coccyoid degenerate forms and coccyoid whole *H. pylori* [3]; moreover, in *Vibrio*-like strains, reversion from dormant coccyoid to bacillary forms has been demonstrated [4]. The interest in the "dormant" coccyoid *H. pylori* is related to the clinical significance of these forms. In fact, this morphology can be generated in vitro with prolonged incubation and also with subinhibiting concentrations of drugs. Therefore, we can hypothesize that with incomplete clearing of the organism, when therapy in *H. pylori*-positive patients is suspended, "stressed" *H. pylori* forms can be generated [5], and this morphology may account for the wide number of relapses.

I suggest that the study of the significance of coccyoid *H. pylori* [6] and, in particular, the quantification of whole strains in this morphology are very important to determine the organism’s mode of transmission. Therefore, I think that Eaton et al. [1] can conclude that coccyoid *H. pylori* does not revert in their animal model but they cannot conclude that with prolonged incubation *H. pylori* has only degenerate forms that cannot infect animal models.

Luigina Cellini

*Istituto di Medicina Sperimentale, Facoltà di Medicina, Università “G. D’Annunzio,” Chieti, Italy*

References


Reply

To the Editor—We would like to thank Dr. Cellini for the opportunity to apologize for failing to cite this author’s work in our original publication. This was an important oversight, and we regret the error. Like Cellini [1], we were surprised by the reported colonization of BALB/c mice by coccyoid forms of *Helicobacter pylori*. This is especially interesting given the acknowledged low and variable colonization of *H. pylori* in mice [2–4] compared with piglets [5,6], puppies [7], and possibly other mammals [8,9]. Cellini suggests that one explanation for the difference between mice and piglets may be the different postinoculation intervals used in the two experiments (2 weeks in piglets vs. 4 weeks in mice). While this is possible, it is our experience that infections in piglets peak 1–2 weeks after inoculation, decrease slightly, and stabilize thereafter. This infection pattern was the reason behind our choice of 1- and 2-week sampling intervals.

Other possible explanations for the different results in the two studies include inoculum preparation, growth conditions, and bacterial strain. In our study, we were careful to use an inoculum dose of coccyoid bacteria that was as close as possible to the dose of bacillary forms. With large numbers of bacteria, it can be difficult to completely ensure that all bacillary forms are absent and we wished to eliminate that possibility as much as possible. Cellini used a much higher dose in a smaller animal, leaving open the possibility that undetected bacillary forms were present. In addition, Cellini and colleagues (see references in [1]) also cultured only 100 \(\mu\)L of the coccyoid sample to ensure absence of bacillary forms, while we cultured 10 mL. While neither method completely assures absence of bacillary forms, we felt that it was important...