We examined pathological changes in the formation of *Helicobacter pylori*-induced gastric lesions in Mongolian gerbils. *H. pylori* (NCTC11637) was orally administered once to the animals and was detected in the gastric mucosa of all gerbils given the bacteria. The number of viable *H. pylori* increased during the initial two weeks and thereafter reached a plateau level. The initial pathological changes were found at one week, ie, edema/congestion and a white viscous substance only in the antrum. At two weeks, superficial damage appeared in the antrum, although inflammatory cell infiltration had not occurred. Gastritis with lymphoid follicles was observed in the antrum and fundus from three weeks. At four weeks, mucosal lesions were detected as a few hemorrhagic spots in the fundus adjacent to the antrum. In the control animals, however, no pathological changes were observed even at four weeks. In the gastric mucosa infected with *H. pylori*, myeloperoxidase activity was negligible at two weeks, but was extremely elevated at four weeks. Similarly, neutrophil chemotactic activity was only slightly increased at two weeks, but was markedly elevated at four weeks. These results indicate that *H. pylori* infection induces initial pathological changes only in the antrum, but mucosal lesions occur in the fundus adjacent to the antrum. Furthermore, it is demonstrated that the initial superficial damage is generated by factors other than chemokines and neutrophil-associated factors, although mucosal inflammation may contribute to the subsequent formation of lesions and ulcers.

**KEY WORDS:** *H. pylori*; Mongolian gerbil; gastric mucosal lesions; inflammation; myeloperoxidase; neutrophil chemotaxis.

It is generally accepted that infection of the human stomach by *Helicobacter pylori* is involved in the occurrence of chronic gastritis and gastric/duodenal ulcers (1, 2). *H. pylori* infection is also associated with high risks of gastric lymphoma and carcinoma (2–4). Thus, eradication therapy has been widely performed for *H. pylori*-infected patients. However, it remains unclear how *H. pylori* infection causes gastric diseases (5). Several researchers have used animals such as piglets (6–8), dogs (9), monkeys (10, 11), germfree mice (12), nude mice (13) and normal mice (14) for studies of *H. pylori* infection. However, the formation of gastric ulcers was only confirmed in piglets (8). Therefore, adequate models involving small animals have been needed in order to develop new therapies and drugs and to elucidate the pathogenesis of *H. pylori*-associated diseases. Recently, it was found that normal Mongolian gerbils can be easily infected with *H. pylori* (15–17). Hirayama et al (17) succeeded in
Fig 1. Infection by H. pylori of the stomachs of Mongolian gerbils. (A) At the indicated times after H. pylori was orally administered once to gerbils, the numbers of viable bacteria in their stomachs were determined by the culture method. After H. pylori was cultured in broth for 10 or 20 passages, H. pylori was orally administered once to gerbils (inset). Four weeks later, the numbers of viable bacteria in their stomachs were determined. Data are presented as means ± SE (N = 8). The numbers in parentheses are the incidences of the detection of H. pylori. (B) H. pylori colonization of the fundic mucosa was detected by immunostaining with anti-H. pylori antibody (four weeks after administration, ×5).