8. Breuillard F, Szapiro E: Cimetidine in acquired tolerance to dinitrochlorobenzene. Lancet 1:726, 1978
9. Böyum A: Isolation of mononuclear cells and granulocytes from human blood. Scand J Clin Lab Invest Suppl 97:77-89, 1968

The authors reply

We appreciate the comments of Kaya and colleagues; however, more information about the inoculum size used, the adequacy of the broth, the response in controls, and the use of any quantitative determination of inhibitor activity is necessary to interpret the data they cite.

The issue remains whether *Helicobacter pylori* is a pathogen or a commensal. At least in the stomach, overwhelming evidence is available in support of a causative role for *H. pylori* in chronic gastritis, and Koch's postulates have been fulfilled.[1-3] The clinical significance of *H. pylori* gastritis, however, remains controversial; clearly, *H. pylori* gastritis is common in totally asymptomatic persons.[4]

A very strong association has been noted between *H. pylori* gastritis and chronic duodenal ulcer disease.[1,2] In their letter, Kaya and co-workers report that cimetidine, which effectively promotes healing of ulcers, did not have antibacterial or phagocytosis-promoting effects. Other investigators have shown that the 90% minimal inhibitory concentration of the H_2 antagonists is more than 1,000 mg/liter.[5] The lack of effect of cimetidine on phagocytic function of macrophages is of interest; although little information has been published on the role of macrophages in *H. pylori* gastritis, they are usually considerably increased in this condition. One study suggested that, in vitro, the speed of ingestion of *H. pylori* by mouse peritoneal macrophages varied with different bacterial strains, but the importance of this finding in vivo is unknown.[6]

Clearly, ulcers heal with H_2-receptor blockers despite the continued presence of *H. pylori*.[7] This result implies that a decrease in gastric acid is sufficient to allow healing of ulcers, and *H. pylori* is not independently capable of inhibiting this process. This finding does not, however, negate a possibly important role of *H. pylori* in duodenal ulcer disease. Indeed, relapse rates for chronic duodenal ulcer disease seem to be substantially less at 1 year in those patients who have had *H. pylori* infection eradicated in comparison with those who have not,[8,9] although further data are needed.

Nicholas J. Talley, M.B., Ph.D.
Roy G. Shorter, M.D.
Sidney F. Phillips, M.D.

REFERENCES

1. Talley NJ: Chronic (nonerosive) gastritis: pathogenesis and management. Dig Dis 7:61-75, 1989
2. Ormand JE, Talley NJ: *Helicobacter pylori*: controversies and an approach to management. Mayo Clin Proc 65:414-426, 1990
3. Ormand JE, Talley NJ, Shorter RG, Carpenter HA, Rouse M, Wilson W, Phillips SF: Prevalence of *Helicobacter pylori* in specific forms of gastritis. Dig Dis Sci (in press)
4. Dooley CP, Cohen H, Fitzgibbons PL, Bauer M, Appleman MD, Perez-Perez GI, Blaser MJ: Prevalence of *Helicobacter pylori* infection and histologic gastritis in asymptomatic persons. N Engl J Med 321:1562-1566, 1989
5. Andreasen JJ, Andersen LP: *In vitro* susceptibility of *Campylobacter pyloridis* to cimetidine, sucralfate, bismuth and sixteen antibiotics. Acta Pathol Microbiol Immunol Scand [B] 95:147-149, 1987
6. Gavinet AM, Megraud F: *In vitro* phagocytosis of *Campylobacter pylori* by macrophages obtained from Biozzi's mice. Proceedings of the first meeting of the European *Campylobacter pylori* Study Group. Bordeaux, France, October 1988, Abstract No. 119
7. Hui W-M, Lam S-K, Chau P-Y, Ho J, Lui I, Lai C-L, Lok AS, Ng M-T: Persistence of *Campylobacter pyloridis* despite healing of duodenal ulcer and improvement of accompanying duodenitis and gastritis. Dig Dis Sci 32:1255-1260, 1987
8. Marshall BJ, Goodwin CS, Warren JR, Murray R, Blincow ED, Blackbourn SJ, Phillips M, Waters TE, Sanderson CR: Prospective double-blind trial of duodenal ulcer relapse after eradication of *Campylobacter pylori*. Lancet 2:1437-1442, 1988
9. Coghlan JG, Gilligan D, Humphries H, McKenna D, Dooley C, Sweeney E, Keane C, O'Morain C: *Campylobacter pylori* and recurrence of duodenal ulcers—a 12-month follow-up study. Lancet 2:1109-1111, 1987