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Mastic Gum Kills *Helicobacter pylori*

To the Editor: Even low doses of mastic gum — 1 mg per day for two weeks — can cure peptic ulcers very rapidly, but the mechanism responsible has not been clear. We have found that mastic is active against *Helicobacter pylori*, which could explain its therapeutic effect in patients with peptic ulcers.

Mastic is a resinous exudate obtained from the stem and the main leaves of *Pistacia lentiscus*. It is used as a food ingredient in the Mediterranean region. Clinically, mastic has been effective in the treatment of benign gastric ulcers[1] and duodenal ulcers.[2] In rats, mastic showed cytoprotective and mild antisecretory properties.[3] We assessed the antibacterial properties of mastic against *H. pylori*.

The *H. pylori* strains NCTC 11637 (a standard reference strain) and six fresh clinical isolates (three were sensitive and three were resistant to metronidazole) were maintained by passage on 7 percent horse chocolate blood agar or in IsoSensitest broth (with 5 percent fetal-calf serum) at 37°C in a microaerobic atmosphere (6 percent oxygen and 5 percent carbon dioxide in nitrogen).

Mastic was prepared as a stock solution in ethanol at a concentration of 50 mg per milliliter and diluted in the broth culture (containing 107 cells of *H. pylori* per milliliter) for a final concentration ranging from 0.0075 to 1.0 mg per milliliter. Ethanol was added to control cultures at appropriate concentrations. The cultures were incubated, 10-μl aliquots were obtained and seeded on agar plates at various times for up to 48 hours, and the minimal bactericidal concentrations (the minimal concentration of drug required to kill 99.9 percent of the organisms in the medium after overnight incubation) were determined.

Mastic killed the *H. pylori* NCTC 11637 strain and the six clinical isolates (reduction in the viable count by a factor of 1000) irrespective of the organism's level of susceptibility to nitroimidazoles. The minimal bactericidal concentration at 24 hours for all strains that were studied was 0.06 mg of the crude mastic per milliliter. At lower concentrations, bacterial growth was still significantly inhibited, with a clear postantibiotic effect even at the lowest concentration used, 0.0075 mg per milliliter. Mastic induced clear ultrastructural changes in the organism, as demonstrated by transmission electron microscopy (data not shown).

These results suggest that mastic has definite antibacterial activity against *H. pylori*. This activity may at least partly explain the anti–peptic-ulcer properties of mastic.[1,2] Examination of the anti–*H. pylori* effect of the various constituents of mastic, which have been recently identified,[4] may pinpoint the active ingredient. Mastic is cheap and widely available in Third World countries; therefore, our data should have important implications for the management of peptic ulcers in developing countries.

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Relation between Chubby Cheeks and Visceral Fat

To the Editor: An upper-body distribution of fat, especially with increased visceral fat, is more predictive of the metabolic complications of obesity than is the degree of overweight.[1] Insulin resistance, type 2 diabetes mellitus, dyslipidemia, and hypertension are associated with upper-body and visceral obesity. We noted that patients with chubby facial cheeks tended to have upper-body obesity and hypothesized that cheek and visceral fat might accumulate in concert. To assess this observation, we measured cheek (buccal), visceral, and abdominal subcutaneous fat in 25 consecutive patients who underwent computed tomographic (CT) scanning of the head and abdomen for clinical purposes within a two-week period.

The patients were being evaluated at the Mayo Clinic for a variety of medical problems, including the evaluation or follow-up of cancer (17 patients), trauma (5 patients), and fever of unknown origin, vascular aneurysms, and nonmalignant masses (1 patient each). None of the patients were seen by us, preventing us from selecting patients whose appearance was consistent with our hypothesis. No patients were taking or had recently taken a glucocorticoid, none had Cushing's syndrome or other disorders of the pituitary or adrenal glands, and none had recently gained or lost a substantial amount of weight. Using the original digital files from the CT scans, we measured the cross-sectional areas of buccal adipose tissue, visceral (intraabdominal) adipose tissue at the L2–3 interspace, and abdominal subcutaneous adipose tissue at the L2–3 interspace.[2] The mean values were 17.7 cm^2 for buccal fat