**ABSTRACT:** Helicobacter are gram-negative, spiral-shaped bacteria. Infection is thought to occur via ingestion. The role of Helicobacter in the pathogenesis of canine and feline gastrointestinal disease is unknown. Many animals harboring the bacteria do not show signs. Diagnostic testing for Helicobacter can be conducted with invasive (gastric biopsy) and noninvasive methods. Many treatment protocols exist, and antibacterial drug resistance is an emerging problem in human medicine. This article provides a comparative review of Helicobacter pathogenesis, diagnostic testing, and treatment between human and veterinary medicine.

Helicobacter spp are gram-negative, spiral-shaped bacteria. At least 13 species have been reported, and most are suspected or proven gastric or hepatic pathogens. Helicobacter spp have been reported in humans (mainly *Helicobacter pylori*), nonhuman primates (*Helicobacter nemestrinae*), cats and dogs (various species, including *Helicobacter felis* and *Helicobacter bizzozeronii*), pigs (*Helicobacter heilmannii*), ferrets (*Helicobacter mustelae*), and cheetahs (*Helicobacter acinonyx*).

In humans, *H. pylori* infection has been associated with chronic gastritis, peptic ulcers, and gastric neoplasia (mucosa-associated lymphoid tissue–type lymphoma and carcinoma). Some humans with *H. pylori* infection develop only mild, asymptomatic gastritis. Whether more severe disease develops is thought to be influenced by individual host factors and pathogenicity of the bacteria involved. The odds of developing symptomatic *H. pylori* infection vary with geographic location and age. Different strains of *H. pylori* have recently been identified. Furthermore, certain *H. pylori* genes have been associated with increased incidence of peptic ulcer and gastric neoplasia formation. Therefore, *H. pylori* should be considered a population of closely related but genetically heterogenous bacteria of differing genotypes and virulence.

In humans, the reservoir for *H. pylori* is the stomach. Risk factors for *H. pylori* infection in humans include age and socioeconomic status, with children and those of low socioeconomic status at greater risk. The role of *Helicobacter* spp in gastrointestinal (GI) disease in dogs and cats is uncertain. It has been known for years that spiral bacteria are commonly present in the stomachs of dogs. The relationship of these organisms to the presence and pathogenesis of gastric disease is not clear.

*H. pylori* transmission is proposed to be fecal–oral, oral–oral, or gastro–oral
Helicobacter spp produce the enzyme urease, which breaks down urea into ammonia and bicarbonate ions. In the stomach, ammonia has a buffering effect that may help Helicobacter colonize mucosa in the normally acidic gastric environment. In addition, ammonia is directly toxic to gastric epithelial cells.¹

### Humans

*H. pylori* infection is associated with increased gastric acid secretion (hyperacidity), which causes inflammation in the gastric antrum (antral gastritis) and duodenal ulceration. It has been proposed that hyperacidity is caused by hypergastrinemia resulting from the inhibition of somatostatin-secreting cells (somatostatin inhibits gastrin release).¹⁴ Hypergastrinemia also increases parietal cell mass through a trophic effect on gastric mucosa. *H. pylori* infection can also be associated with lack of gastric acid (achlorhydria). This is thought to occur when *H. pylori* causes mucosal atrophy in the gastric fundus and body or inhibits functioning of the parietal cells.²,¹⁵ Chronic gastric inflammation may progress to chronic atrophic gastritis and intestinal metaplasia, which are precancerous conditions.⁷

### Dogs and Cats

It appears that *Helicobacter* infection does not significantly alter gastric acid secretion in dogs. Study of naturally infected dogs and cats has shown that *Helicobacter* predominantly colonizes the gastric fundus and cardia and is associated with mild to moderate mononuclear cell inflammation.¹⁶ In a study of beagles infected with *H. felis*– and *H. bizzozeronii*–like organisms, Simpson et al.¹⁶ concluded that acid secretion was not markedly perturbed by infection and that treatment temporarily suppressed but did not eradicate the bacteria. Infected dogs showed no signs and had mild gastritis histologically before and after treatment (mild gastric inflammation was also found in the uninfected control dogs of this study). No correlation was identified between the degree of inflammation and degree of bacterial colonization.² Happonen et al.¹⁷ found that successful treatment of *Helicobacter* in pet dogs did not change gastric histology and that mild chronic gastritis persisted following treatment, even in dogs that tested negative to *Helicobacter* infection, based on repeat endoscopic biopsy, brush cytology, and urease testing.

### DIAGNOSIS

#### Humans

Diagnostic tests for *Helicobacter* can be divided into invasive (those requiring a gastric mucosal biopsy specimen) and noninvasive methods. *H. pylori* may exhibit a patchy distribution in gastric mucosa and may cause atrophic gastritis (with resultant low bacterial numbers in the gastric mucosa). Both factors may limit sensitivity of biopsy-based diagnostic tests.⁸

Of the invasive methods, culture of a gastric biopsy specimen is considered the gold standard.⁸ Unfortunately, organism characteristics make culture difficult,¹⁸ and false-negative results can occur because of loss of bacterial viability if samples are not cultured quickly (within 6 to 24 hours, depending on storage temperature).¹⁹ Culture is considered 100% specific, but sensitivity varies widely with laboratory expertise.¹⁹ In experienced hands, sensitivity of culture is reportedly greater than 90%.¹⁸

Other invasive tests include rapid urease testing, direct observation of *Helicobacter* organisms via histopathology or electron microscopy, and polymerase chain reaction (PCR) testing. Only culture, electron microscopy, and PCR can be used to identify the particular *Helicobacter* sp present.

Rapid urease tests identify *Helicobacter* by detecting urease activity.¹⁹ A gastric mucosal biopsy specimen should be embedded in urea containing gel medium. Urease from the *Helicobacter* (if present) hydrolyzes urea, increasing pH and changing the color of the medium. A positive result is a color change from orange–red to bright pink. Rapid urease tests are commercially available and can be used with endoscopic biopsy samples. Urease testing should be conducted on biopsy specimens within 24 hours, or false-negative results may occur.¹⁸ False-negative results may also occur following treatment because of low organism numbers in the biopsy sample¹⁹ and following the use of antisecretory drugs.¹⁰ False-positive results may occur with artificial pH increases because of contamination of the sample with blood or urease-producing bacteria from the oral cavity.¹⁸ The sensitivity and specificity of rapid urease tests are reportedly very good (88% to 100%),¹⁹,²¹ except in patients with bleeding duodenal ulcers.²²

Direct observation of *Helicobacter* organisms in biopsy specimens usually requires special stains (e.g., Giemsa, Warthin-Starry, Gent’s, alcian yellow- tolui-
dine blue). *H. pylori* is typically not visualized with the commonly used hematoxylin and eosin stain. Current standards in human medicine dictate that an estimate of *H. pylori* density, activity and grade of gastritis, and comments on the presence of atrophic gastritis or intestinal metaplasia should be provided when histopathologic examination is made. The specificity of histopathology is 100%, and sensitivities of greater than 90% have been reported. Immunochemical staining and electron microscopy may also be used to evaluate gastric biopsy specimens.

PCR allows identification of the particular *H. pylori* sp or strain present and can be conducted using gastric biopsies, gastric juice, dental plaque, or feces. Sensitivity varies with the primer used but is considered high. In addition, PCR allows identification of specific *H. pylori* genes associated with an increased incidence of peptic ulcer or cancer (so-called virulence factors).

Noninvasive diagnostic methods include urea breath testing, a stool antigen test, and serum antibody testing. Although consensus exists among gastroenterologists that noninvasive testing should be conducted first in patients suspected of *H. pylori* infection, agreement is lacking regarding which noninvasive test is best. Urea breath testing (which detects urease activity) and the stool antigen test (which measures *H. pylori* antigen) are direct tests that detect active infection. Serum antibody tests are indirect and cannot distinguish between actively infected patients and those that were previously infected. Direct testing is thus preferred. However, serum antibody testing is currently the most commonly used screening method because of convenience, availability, and low cost.

Like the rapid urea test, urea breath testing finds *Helicobacter* by identifying urease activity. The patient swallows labeled (with nonradioactive carbon 13 or radioactive carbon 14) urea. If *Helicobacter* is present, urease hydrolyzes the labeled urea and the marker (carbon-labeled bicarbonate) is detected in the breath after 30 to 60 minutes. A test meal is given to delay gastric emptying. Antisecretory drugs increase gastric pH and decrease urease activity; thus test results may be false-negative immediately after treatment and must be interpreted in relation to treatment. The current recommendation in human medicine is to wait 4 weeks following cessation of therapy before conducting a follow-up urea breath test, although research has demonstrated that the effect of antisecretory drugs on the urea breath test can resolve as early as 5 days after cessation of treatment. Sensitivity and specificity of urea breath testing are high (greater than 95%), and the urea breath test is considered a reliable, noninvasive way of documenting eradication after treatment. Carbon-labeled bicarbonate can also be detected in serum, but FDA approval of this as a diagnostic test for *Helicobacter* is pending.

The stool antigen test, an enzyme immunoassay test for detecting *H. pylori* antigen, is the newest noninvasive technique for diagnosing *Helicobacter* infection in humans. Initial test kits employed polyclonal antibodies to *H. pylori*, but a newer test using monoclonal antibodies has been developed. Based on a review of 44 studies encompassing 4769 untreated patients, Gisbert and Pajares found that sensitivity and specificity for *H. pylori* stool antigen testing were high (greater than 92%). The optimal time for using stool antigen testing after treatment to check for eradication is uncertain. The accuracy of this test in patients with GI bleeding is uncertain. It is also uncertain whether cross-reactivity with other *Helicobacter* spp exists.

Serology identifies antibodies to *H. pylori*, which can be detectable for more than 1 year following infection. Three types of antibody tests are available: quantitative serum ELISA, Western blot test using serum, and qualitative rapid whole blood tests (in-office, rapid latex agglutination or flow-through, membrane-based enzyme immunoassays). Serum ELISA testing is the most commonly used method and can detect IgG and IgA. The serum IgG ELISA is considered most sensitive, although sensitivity depends on characteristics of the population tested and the particular *H. pylori* antigen preparation used to make the test kit. Herbrink and van Doorn reported that sensitivity and specificity of commercial ELISA assays vary 60% to 100%, and most had values over 85%. Western blot testing of serum permits more detailed analysis of the patient antibody profile but has limited usefulness because of labor-intensive methodology. Variable sensitivity has been reported for in-office, rapid, whole blood tests designed for use in primary care clinics.

Antibodies (IgG) to *H. pylori* can also be detected in saliva, but this method had lower sensitivity and specificity (81% and 73%, respectively) than serum antibody testing (90% sensitivity, 78% specificity) in a recent multicenter study of 213 dyspeptic patients. IgG against *H. pylori* has also been detected in urine, and a urine-based ELISA test recently validated in Japan had an accuracy comparable to that of the serum ELISA test.

**Dogs and Cats**

Noninvasive test methods for detecting *Helicobacter* (e.g., urea breath testing, antibody testing, stool antigen testing) are not routinely available for dogs and cats. Urease testing (breath and blood) has been inves-
tigated in dogs but is not widely available.\textsuperscript{25,29} Antibody testing in dogs and cats is potentially more difficult than in humans because of the variety of \textit{Helicobacter} spp infecting dogs and cats. Nonetheless, antibody testing is being investigated, and infected animals are known to develop antibodies.\textsuperscript{30,31} Theoretically, stool antigen testing could be useful in \textit{H. pylori}–infected cats (or in animals infected with \textit{Helicobacter} spp that have antigenic homology and thus cross-reactivity with \textit{H. pylori}). This has not been investigated.

The only way to confirm the presence of \textit{Helicobacter} in dogs and cats is with the invasive methods already discussed. Endoscopically obtained gastric mucosal biopsies are commonly used, and direct observation of organisms (via histologic examination of biopsies or cytologic examination of brush cytology specimens) and rapid urease testing are common methods of identifying \textit{Helicobacter}. Because \textit{Helicobacter} distribution in the stomach may be patchy, evaluation of multiple biopsies and anatomic locations (i.e., cardia, fundus, antrum) is recommended.\textsuperscript{24} Figures 1 through 3 show photomicrographs of \textit{Helicobacter} spp in feline gastric mucosa. Unfortunately, the lack of noninvasive diagnostic testing for \textit{Helicobacter} in veterinary medicine makes response to treatment difficult to assess. Repeat endoscopy or biopsy is required, which is expensive and unappealing to many pet owners. However, an advantage of follow-up endoscopy over noninvasive testing is the opportunity to reassess gastric morphologic changes.

**TREATMENT**

**Humans**

Eradiication therapy for \textit{Helicobacter} is recommended for humans with symptomatic \textit{H. pylori} infection. Treatment guidelines exist for the various subcategories of GI disease caused by \textit{H. pylori} in humans.\textsuperscript{7} In general, eradication therapy consists of antibiotic drugs in conjunction with antisecretory drugs. Some gastroprotective drugs have also been reported to have antimicrobial activity against \textit{H. pylori}.\textsuperscript{32}

Experience in humans has revealed that multiple drugs are needed to cure \textit{H. pylori} infection. The most successful treatment regimens consist of at least two antibiotics combined with bismuth and/or an antisecretory drug. Many combination drug regimens exist in human medicine. Treatment duration is 1 to 2 weeks; 10- and 14-day treatment regimens have better cure rates and are considered superior to 7-day regimens. Currently recommended antimicrobials include amoxicillin, tetracycline, metronidazole, clarithromycin, and bismuth (Table 1). Antisecretory therapy accompanies antibiotic therapy because eradication rates increase...
when both are used together. Furthermore, the antimicrobial activity of amoxicillin and clarithromycin is better at a higher pH. Both H₂-receptor antagonists and proton-pump inhibitors can be used for antisecretory therapy, but proton-pump inhibitors are preferred because they suppress acid production more reliably, provide more rapid ulcer healing, and have anti–H. pylori activity in vitro. Classic triple therapy in humans consists of bismuth, metronidazole, and tetracycline. New triple therapies refers to combinations of antibiotics and a proton-pump inhibitor. Quadruple therapy refers to classic triple therapy that has been modified by increasing the dose of metronidazole and adding a proton-pump inhibitor.\textsuperscript{32}

Antibiotic resistance of H. pylori is a problem in human medicine and has been reported for clarithromycin, metronidazole, amoxicillin, and tetracycline. Interestingly, combination therapies involving metronidazole have been used successfully despite apparent metronidazole resistance in vitro. Treatment failure in humans is caused by antibiotic resistance or insufficient suppression of gastric acid production.\textsuperscript{32}

**Dogs and Cats**

Treating Helicobacter infection in dogs and cats is controversial. Whether treatment is needed in all cases and which drugs are preferred is not clear. Efficacy of treatment is also unclear, and it is unknown whether drug resistance is a problem in veterinary patients. Recent studies suggest that treatment might only suppress infection but not eradicate it.\textsuperscript{2,17,25} More reports of post-treatment follow-up assessing bacterial status and GI

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**Table 1. Drugs Used To Treat H. pylori Infection in Humans\textsuperscript{32, a}**

<table>
<thead>
<tr>
<th>Drug Class</th>
<th>Recommended Drug(s)</th>
<th>Mechanism</th>
<th>Resistance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Penicillins</td>
<td>Amoxicillin</td>
<td>Inhibits bacterial wall synthesis</td>
<td>Not common; does produce cross-resistance to other penicillins</td>
</tr>
<tr>
<td>Tetracyclines\textsuperscript{a}</td>
<td>Tetracycline</td>
<td>Inhibits bacterial protein synthesis by binding the 30S ribosomal subunit</td>
<td>Not common</td>
</tr>
<tr>
<td></td>
<td>Oxytetracycline</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nitroimidazoles</td>
<td>Metronidazole</td>
<td>Damages bacterial DNA</td>
<td>Common in countries where it is used for diarrhea, amoebiasis, or trichomoniasis</td>
</tr>
<tr>
<td>Macrolides</td>
<td>Clarithromycin</td>
<td>Inhibits bacterial protein synthesis by binding the 50S ribosomal subunit</td>
<td>Increasing; may limit usefulness in the future</td>
</tr>
<tr>
<td>Bismuth salts</td>
<td>Bismuth subsalicylate</td>
<td>Disrupts bacterial cell walls</td>
<td>Not reported</td>
</tr>
<tr>
<td></td>
<td>Bismuth subcitrate</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Ranitidine–bismuth</td>
<td>Combination of bismuth and the H₂ antagonist ranitidine; an advantage is that bismuth is soluble in acid</td>
<td>Not reported</td>
</tr>
<tr>
<td></td>
<td>citrate</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Proton-pump inhibitors</td>
<td>Omeprazole</td>
<td>Suppresses gastric acid production via inhibition of the sodium-potassium-ATPase pump</td>
<td>Does not apply</td>
</tr>
<tr>
<td></td>
<td>Lansoprazole</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Pantoprazole</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Rabeprazole</td>
<td></td>
<td></td>
</tr>
<tr>
<td>H₂-receptor antagonists</td>
<td>Cimetidine</td>
<td>Suppresses gastric acid production by competitively inhibiting histamine</td>
<td>Does not apply</td>
</tr>
<tr>
<td></td>
<td>Famotidine</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Nizatidine</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Ranitidine</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Roxatidine</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

\textsuperscript{a}Doxycycline is reportedly ineffective for treating H. pylori.\textsuperscript{24}
changes are needed for naturally acquired clinical cases. Treating *Heli-
cohacter* infection in a dog or cat should be considered if all of the fol-
lowing are true:

- The animal has clinical signs of gastritis.
- The animal has histologic evi-
dence of gastritis.
- *Helicobacter* is identified.
- There is no other apparent cause of the gastritis.

A thorough investigation to rule out underlying GI disease (e.g., food intolerance/hypersensitivity, parasitism, inflammatory bowel disease, neoplasia) should be undertaken before treating *Helicobacter* infection. Even with such a workup, it may be difficult to know whether all GI inflammation is caused by *Helicobacter* infection alone or whether underlying inflammatory disease is present. This is especially true for patients in which inflammation persists following treatment and apparent *Helicobacter* eradication.

Treatment protocols in dogs and cats have been adapted from human medicine and include various combinations of antibiotic and antisecretory therapy. Traditional veterinary triple therapy consists of metronidazole, amoxicillin, and bismuth subsalicylate and has been used as initial treatment for *Helicobacter* infection in dogs and cats. Other antimicrobial drugs that have been used in animals in which traditional triple therapy fails include tetracycline and clarithromycin. Antisecretory drugs that have been used include H$_2$ antagonists (e.g., cimetidine, ranitidine, famotidine) and proton-pump inhibitors (omepra-
zole). The synthetic prostaglandin analogue misopros-
9 tol has also been used. Veterinary drug protocols and dosages are summarized on this page (see box and Table 2). Treatment is typically administered for 2 to 4 weeks, but the optimal duration of treatment is unknown. Bismuth subsalicylate should be adminis-
tered cautiously or not at all in cats because of their innate salicylate sensitivity. I have seen clinical gastritis resolve following 4 weeks of treatment with traditional veterinary triple therapy (in dogs) or a combination of amoxicillin, metronidazole, and famotidine (in dogs and cats).

### Table 2. Drugs Used to Treat *Helicobacter* Infection in Dogs and Cats

<table>
<thead>
<tr>
<th>Drug</th>
<th>Dosage$^{a,b}$</th>
<th>Study</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Antimicrobial</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Amoxicillin</td>
<td>15–20 mg/kg PO q8h</td>
<td>Fox$^{13,33}$</td>
</tr>
<tr>
<td>Clarithromycin</td>
<td>7.5 mg/kg PO q12h</td>
<td>Fox$^{13}$</td>
</tr>
<tr>
<td>Metronidazole</td>
<td>10–20 mg/kg PO q12h</td>
<td>Fox$^{13,33}$</td>
</tr>
<tr>
<td>Tetracycline</td>
<td>20 mg/kg PO q8–12h</td>
<td>Fox$^{13}$, Harponen et al.$^{17}$</td>
</tr>
<tr>
<td><strong>Antisecretory</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cimetidine</td>
<td>5–10 mg/kg PO or IV q8h</td>
<td>Fox$^{13}$</td>
</tr>
<tr>
<td>Famotidine</td>
<td>0.5 mg/kg PO q12h</td>
<td>Simpson et al.$^{2}$, Cornetta et al.$^{25}$</td>
</tr>
<tr>
<td>Ranitidine</td>
<td>1–2 mg/kg PO or IV q12h; 2.5 mg/kg IV or 3.5 mg/kg PO q8h (cats)</td>
<td>Fox$^{13}$</td>
</tr>
<tr>
<td>Omeprazole</td>
<td>0.5–1 mg/kg/day PO (maximum dose in dogs, 20 mg)$^3$</td>
<td>Fox$^{13,33}$</td>
</tr>
<tr>
<td><strong>Other</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Misoprostol</td>
<td>1–5 µg/kg PO q8h (dogs)</td>
<td>Fox$^{13}$</td>
</tr>
<tr>
<td>Bismuth subsalicylate$^a$</td>
<td>0.5–2 ml/kg (dogs) or 0.5–1 ml/kg (cats) PO q6h</td>
<td>Fox$^{13,33}$</td>
</tr>
<tr>
<td>Bismuth subcitrate</td>
<td>6 mg/kg PO q12h</td>
<td>Fox$^{13,33}$</td>
</tr>
<tr>
<td>Sucralfate</td>
<td>0.5–1 g PO q2–4h</td>
<td>Hall$^1$</td>
</tr>
</tbody>
</table>

$^a$Treatment duration is typically 2 to 4 weeks.

$^b$Dosages can be used in both dogs and cats unless specified otherwise.

$^c$Bismuth subsalicylate (Pepto-Bismol™, Procter & Gamble). Use with caution in cats because of their sensitivity to salicylate.

### Suggested Drug Combinations for Treatment of *Helicobacter* Infection

- **Triple therapy (amoxicillin + metronidazole + bismuth)** Fox$^{13,33}$
- **Tetracycline + metronidazole + bismuth** Fox$^{13}$
- **Clarithromycin + metronidazole + bismuth** Fox$^{13}$
- **Amoxicillin + metronidazole + one of the following:** Fox$^{13}$, Cornetta et al.$^{25}$
  - cimetidine, famotidine, ranitidine, omeprazole, or misoprostol
- **Amoxicillin + metronidazole + sucralfate** Hall$^1$
- **Tetracycline + omeprazole** Happonen et al.$^{17}$
- **Amoxicillin + clarithromycin + omeprazole** Hall$^1$
Current Knowledge of the Role of Helicobacter in Dogs and Cats

What We Do Know

Many dogs and cats, healthy and sick, are infected with Helicobacter-like organisms.

Most infected animals are asymptomatic. Gastric acid secretion may not be significantly affected. 2

Helicobacter infection can be associated with lymphocytic-plasmacytic gastritis and proliferation of lymphoid follicles. 1 Degeneration of gastric glands may occur. Infected animals can develop antibodies (seroconvert). 16

Peptic ulcers and gastric neoplasia as a result of Helicobacter infection have not been reported in dogs and cats. 10

Treatment may not eradicate the bacteria, and subclinical gastritis may persist following treatment. 2,25,35

What We Do Not Know

What are the factors that control the interaction between host and Helicobacter and the development of clinical gastritis in dogs and cats? Recent studies have not found a correlation between the degree of colonization and the degree of gastric inflammation in dogs. 2,35 Is there a difference between the young, otherwise healthy laboratory animals used in many studies and the pet population? 26

Is treatment always indicated when Helicobacter is identified? Based on what is known, the answer may be “no” for many dogs and cats. 6 When should infection be considered significant? Are all Helicobacter spp equally significant in the pathogenesis of canine and feline GI disease?

Can treatment eradicate the bacteria in dogs and cats? Does it merely suppress infection, rendering Helicobacter undetectable? 17

How long should treatment last?


Looking to the Future

Much is yet to be learned regarding the role of Helicobacter infection in dogs and cats (see box above). Further research is desperately needed. A systematic approach to diagnosis and treatment, including post-treatment assessment, is vital to furthering our understanding of this infection in the pathogenesis of canine and feline GI disease. PCR may prove helpful in determining the particular Helicobacter sp present and whether treatment truly eradicates or merely suppresses infection. Without such information, it is impossible to know whether apparent treatment failures and relapses are caused by persistence of Helicobacter infection, reinfection, or the presence of underlying GI disease. Development of noninvasive diagnostic tests appropriate for veterinary patients would also help improve our understanding of Helicobacter infection in dogs and cats.

Acknowledgment

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References


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ARTICLE #3 CE TEST
The article you have read qualifies for 1.5 contact hours of Continuing Education Credit from the Auburn University College of Veterinary Medicine. Choose the best answer to each of the following questions; then mark your answers on the postage-paid envelope inserted in Compendium.

1. In humans, the reservoir for H. pylori is ________, and transmission is thought to be ________.
   a. dental tartar; oral–oral or fecal–oral
   b. saliva; oral–oral or gastro–oral
   c. the stomach; oral–oral, fecal–oral, or gastro–oral
   d. the proximal small intestine; fecal–oral or gastro–oral

2. H. pylori causes gastric disease by
   a. producing urease, which causes ammonia formation.
   b. causing gastric hyperacidity.
   c. causing gastric mucosal atrophy.
   d. all of the above

3. In dogs and cats, Helicobacter infection may be associated with
   a. lymphocytic-plasmacytic gastritis.
   b. prominent lymphoid follicles.
   c. gastric lymphoma.
   d. a and b

4. The gold standard diagnostic test for Helicobacter is ________, but it is often not conducted because of ________.
   a. culture; the difficulty in culturing the organism
   b. urease testing; the need for gastric biopsy
   c. the stool antigen test; limited availability
   d. endoscopic biopsy; the expense

5. Which diagnostic test can be used to identify particular species of Helicobacter?
   a. urease testing
   b. PCR
   c. stool antigen test
   d. direct examination of organisms (biopsy)

6. Rapid urease tests work by detecting
   a. labeled bicarbonate.
   b. anti–H. pylori antibodies.
   c. an increase in pH due to urease activity.
   d. H. pylori antigen.

7. Detecting spiral bacteria in gastric biopsy specimens is not possible without
   a. full-thickness biopsies.
   b. electron microscopy.
   c. special staining.
   d. immunohistochemistry.

8. Treatment failure of Helicobacter infection can be due to
   a. antimicrobial resistance.
   b. inadequate suppression of gastric acid secretion.
   c. recrudescence of infection.
   d. all of the above

9. Bismuth is effective for treating Helicobacter infection because
   a. it reduces gastric acid.
   b. it disrupts bacterial cell walls, killing the bacteria.
   c. it comes combined with ranitidine.
   d. the salicylate component is antiinflammatory.

10. Which antibiotic(s) is useful for treating H. pylori?
    a. amoxicillin  
    b. metronidazole
    c. clarithromycin
    d. all of the above