

ACTA SCIENTIFIC MICROBIOLOGY (ISSN: 2581-3226)

Volume 4 Issue 3 March 2021

Review Article

Helicobacter pylori Treatment

Abdulaziz Radhi S ALjohni^{1*}, Bassam A Aljohny², Sultan Saud Alahmad³, Abdulaziz M Hakami⁴, Omar Alfaroq⁵ and NA Al-Tayyar⁶

¹Department of Laboratory at King Fahad Hospital, Medina, Saudi Arabia

²Department of Biological Sciences, Faculty of Science, King Abdulaziz University, Jeddah, Saudi Arabia

³Head of Public Health Department at Ohud Hospital Family Medicine Registrar, Medina, Saudi Arabia

⁴Center for Disease Prevention and Control (NCDC), Jazan, Saudi Arabia

*Corresponding Author: Abdulaziz Radhi S ALjohni, Department of Laboratory at King Fahad Hospital, Medina, Saudi Arabia.

Received: February 08, 2021

Published: February 25, 2021

© All rights are reserved by Abdulaziz Radhi

S ALjohni., et al.

Abstract

H. pylori the *Helicobacter* genus, is found in over 80% of cases of gastric cancer in developed and emerging nations. *H. pylori* is gram-negative and is responsible for gastroduodenal diseases as the recognized major human bacterial pathogen. It will remain in the stomach for life until acquired and colonized. Acute and chronic gastritis, peptic ulcer disease, gastric adenocarcinoma and lymphoid tissue tumor combined with mucosa are found in a limited population of carriers (MALTA). In certain instances, H. The *pylori* infection is absolutely asymptomatic or has signs that are nearly imperceptible. *H. pylori* are different depending on the form of media, blunted/rounded ends in gastric biopsy specimens, but in fresh culture on agar medium they are rod-like and spiral shape when coccoid is on prolonged culture.

Infection therapy depends on a mixture of antimicrobial agents and antisecretory agents, and the bactericidal activity of antimicrobial agents includes an elevation of gastric pH by antisecretory agents. Alternatively, although the mechanism of action is not yet apparent, to enhance eradication of H, phytomedicines and probiotics have been used *H. pylori*. The effect of antimicrobials and antisecretory agents is not only based on their pharmacological action, but also on their pharmacokinetic properties. Several antimicrobial agents have been used for H, including amoxicillin, clarithromycin, levofloxacin, metronidazole, tetracycline, rifabutin and compounds containing bismuth *H. pylori*.

Keywords: H. pylori (Helicobacter pylori) Therapy; Infection

Introduction

H. pylori, an agent that tumors and (once mistaken for a bacteria), first identified at *Campylobacter pyloridis*, was first identified

in humans and cultured by Marshall and Warren [1]. It is a gramnegative bacterium, microaerophilic, spiral-shaped, with multiple polar mobility flagels. Only at a periplasmic pH of 4.0 - 8.5 can it

⁵Department of Laboratory at King Fahad Hospital, Medina, Saudi Arabia

⁶General Administration of Education in Jeddah Governorate, Saudi Arabia

survive and can only develop at a periplasmic pH of 6.0 - 8.5. One well-recognized metabolic feature of *Hemophilus*, ill is its capacity to generate urease, a digestive enzyme that can hydrolyze gastric urea to liberate ammonia, neutralizes the gastric acid and raises the periplasmic pH to 4 - 6, thereby preserving H. Gastric acid from H. pylori [2]. The approximate routes of H. The propagation of pylori remains unknown. Epidemiological studies have shown, however, that food exposure to polluted water or soil can increase the risk of *H. pylori* infection indicates that the most possible route for his person-to-person transmission by oral, fecal-oral, or gastro-oral contact. Infection of *H. pylori* improvements in health and working environments are also essential factors in reducing the incidence of infection. H. pylori has affected more than 50 percent of the world's population. Pylori and the prevalence of infection in developed countries in adults over 50 years of age is more than 80%. Generally, contaminated persons develop H. pylori and grow up with the infection before 10 years of age [3]. In Asia, the H. pylori infection varies in different countries, with a reported total seroprevalence incidence of about 31% in Singapore, 36% in Malaysia, 39% in Japan, 55% in Taiwan, 57% in Thailand, 58% in China, 60% in South Korea, 75% in Vietnam, 79% in India and 92% in Bangladesh [4]. Gastrointestinal disorders, including gastric inflammation, peptic ulcer, gastric cancer and gastric mucosa-related lymphoid tissue lymphoma, are closely associated with *pylori* infection. It has been listed as a category 1 carcinogen by the International Agency for Research on Cancer (IARC) since 1994 [5].

Infection

Any of the disorder with H. Just about 30% - 70% of all humans are contaminated with *H. pylori* bacteria. The predominance of *H. pylori* are considered to be triggered by economic circumstances of a region [6]. The predominance of older people over younger ones is rampant in many countries across the world. The pollution is often spread within families and in early childhood. It is more possible the transfer of (bad) content from individual to individual transpires without individual interfering goods (such as water). At this time, there is no be proved zoonotic transmission, even though H. Uncontrollable ulcers can be observed in a few nonhuman primates and occasionally in other species. *Pyloric* disease is a condition in people, but childhood cannot be easy and accidental treatment happens [9]. Antibiotics were also provided to avoid the secondary infections.

Epidemiology of H. pylori

About half of the world's people was infected with H. pylori, which is acquired almost constantly during the first 5 years of life. The prevalence rates vary from 1.2 percent to 12.2 percent throughout the created universe [10]. The rates of predominance are even higher in developed countries. Serological rates of predominance of H. pylori was 15% and 46% among Gambian children aged less than 20 months and 40 - 60 months, respectively and 45% among Indian children. In Bolivia and the frozen north, the seroprevalence was 70 percent and 69 percent individually at the age of 9 a long time [11]. Seroprevalence was found to be 69.7 percent in pre-school children in Brazil [12] an age-related rise in H predominance. A few free thoughts about the planet [13] were watched by *pylori*, irrespective of the nation's financial situation. Components which are inclined towards H. pylori disorder has been analyzed in depth in infants. The economic position was the most inclining variables distinguished in these ponders [14]. Pyloric to those living in swarmed homes, pylori disease is more notable. McCallion., et al. found in their study that the relation between social lessons and H. After modification, pylori are immaterial for family thickness and bed-sharing between an infant and an adult. This observation indicates that it applies to the safeguarding of H. The social lesson of pylori disease was to serve as an intermediate degree for family circumstances and hones that increase the spread of the life form from polluted to uninfected subjects [15]. Improvements in living conditions have contributed to a marked decline in H. Transmission of H. pylori.

Diagnostic

H. pylori in the domestic station, *pylori* was based on example biopsy software. Ultrasound and magnetic resonance scans are more than favorable to pediatric patients Regardless of the efficacy of non-invasive studies, they do not determine an option of damage minimization *pylori* in adolescents [16]. Testing of *H. pylori* antigens in stools has appeared promising comes about in grown-ups for the noninvasive determination of gastric disease employing a commercially accessible pack [17].

The assesses for *H. pylori* antigens in feces are shown to be accurate for use in verifying the destruction treatment victory. Be that as it may, patients may wait too long to gather examples of stools. Refrigerated stools are more difficult to test during expan-

sion. Sometime recently, additional pediatric ponders evaluating the accuracy of stool antigen testing for both initial conclusion and post-treatment follow-up are required, especially suggestions can be considered [18]. H. Cost to treat cystathionine last resistant illness nearly continually actuates a specific systemic resistance reaction which may indicate the antibodies produced at the gastric mucosal stage, while as it were unexpectedly 2% patients fell flat to seroconvert. For this test, a light was used to show how fast this pollution could take place, so it occurred rapidly when this was exposed. a bacteria named [18,19].

By the way, the necessary response is to conform with the auto-immunity display seen in the auto-antigen that is tainting the host. This decision was one of the principal to be related to the position of H. this bacteria. Without a question, the process of freezing and staining the muscle tissue was used as basis for this analysis. bacteria were being hunted for and were eventually being perfected. In certain environments, it is usually decided that X is perhaps one of the utmost widely used, at the very least in countries where endoscopy is very common [20].

Helicobacter pylori therapies

The number of antibiotics used in this field is very high. eradication of *pyloric*.

Amoxicillin is the antibiotic developed by the mold Penicillium chrysogenum. It prevents the synthesis of the bacterial wall, which restricts the bacteria 's growth and makes the body remove it. The bacterial antibacterial activity is time dependent, such that it reaches a sufficient and maintained minimum inhibitive concentration (MIC) when it is administered three or four times daily. Two times a day dosing corresponds with this medication's pharmacokinetic properties such as its short half-life. A standard routine goes like this: 3 to 4 times a day, it should be taken. It can be taken to consistently sustain treatment levels. the correct approaches to handle H. are [21] plowing the stomach can result in very rare examples of this antibiotic. In Latin America, the prevalence of rheumatoid arthritis is 4 percent. In Columbia, the rate is less than 2 percent. When taken as a drug, it is an exceptional medicine and can also be used in second line treatments after one scheme in which it has been used fails [21].

Clarithromycin, a macrolide that binds to the 50S unit of the bacterial ribosome, has been used in combination with other anti-

bacterial agents in multi-drug therapy (MDR) since the early 1990s. Assuming that this is the case, it also means that it has a half-life of 5 hours (strictly speaking, the half-life is technically just 2.5 hours, but because the duration is under 2 hours, there is no problem) [22]. Since it has been commonly recommended for various common diseases, H. the bacteria has established an alarming tolerance to it everywhere. The H. is a very common bacterium in Japan. The resistance rate goes up from 1.8% in 1996 to 27.1% in 2008 while in the China was 14.8% in 2000 and 52.6% in 2014 and over the same time those in the EU declined from 8.2% to 10.2%. In Latin America, the average resistance (the number of times people avoid a certain disease) was 12% before 2011 and in 2017 it is now 25% in Colombia. I totally identify with you. In 2017, the World Health Organisation (WHO) has researched the antimicrobial tolerance of Helicobacter. The world health organisation includes pylori in its list of 16 microorganisms that endanger mankind and is focused on finding a way to remove H. pylori [24].

Metronidazole is the only medication approved for this reason which ruptures the double-chain of bacteria DNA and has a plasma half-life of close to 8 hours so it can be administered 2 or 3 times a day. This drug has raised a lot of controversy, particularly as to its possible side effects and efficacy. This experiment has the average resistance on the continent of 53% of the other societies we have tested. Colombia has the highest recorded resistance of 83%. It is the only drug whose in vitro resistance can be reversed by increasing the dosage and length of therapy. In quadruple treatment, 500 mg 4 times a day or 400 mg 4 times a day for 14 days reaches a 92% effectiveness rate at even with resistant strains [25]. The outcome where there is no resistance to the drug will be the same as when there is a resistance. The mixture of metronidazole, trimethoprim, chloramphenicol, and chlorhexidine is most successful in treating food poisoning induced by trimethoprim resistant bacteria in regions where the possibility of resistance is critical [26].

Quinolones serves to induce breaks in the bacterial DNA, thus altering the bacteria's ability to survive. Their bactericidal effect is marginally more effective than merely adding the bacteria into an elixir as long as the patient takes the elixir on a daily basis. Levo-floxacin [26] the most scientifically applicable and frequently used quinolone, has been typically used as the first line treatment for infections of parenchyma and for certain cases of penicillin allergies and when antibodies to first-choice medications are high. As the

case of clarithromycin resistance, large use of antibiotics to treat other diseases particularly upper respiratory tract infections has led to development of drug resistance. over the whole body. Overall, the resistance ranges between twelve and fifteen percent in Europe, but in Colombia it is a staggering twenty-seven percent [26].

Rifabutin, the rifampicin metabolite, is similar to rifamycin. The medicine has been developed over twenty years ago and has been used to cure H. pylori infection on a last-ditch basis there are several ways to get rid of the infection [27]. Including rescue family planning drugs, it's bioavailability is poor, and it has a wide amount of delivery. It has a larger intracellular membrane permeability potential than rifampicin, which presumably occurs because it is more lipophilic than rifampicin. If there is a DNA sequence homologous to itself, host cell DNA polymerase is inhibited by the antipolymerase, e.g., Bacillus caucasinosporus NLS63 gene [27]. The H is acting strongly. Just one in one hundred people report a sideeffect from taking this drug, but their disadvantages include high prices, a lack of convenient access in many areas, and unpleasant side-effects in more than twenty percent of patients. The most dreaded side-effect of thalidomide is myelotoxicity, which happens when dosages are greater than 600 mg a day or more and which vanish following discontinuation [28].

Furazolidone, a wide range nitrofurans antimicrobial, protects the stomach, is very good at destroying a number of bacteria, and is highly powerful when it comes to heat. A lack of availability is the drawback [29]. Originally used for the treatment of diarrhea in gastroenterology, showed effectiveness against *H. pylori*, especially when bismuth is used with it. Nonetheless, as it combines with soy derivatives and mature cheeses, it also causes harmful effects. Side effects vary from minor conditions such as nausea and emesis, to extreme cases such as epilepsy and hypertension. In China, Iran and Colombia, this drug has been extensively used in care schemes. The resistance recorded in China is 0% to 1%, while in Brazil it is 3% on average [30].

Helicobacter pylori alternative treatments

Honey is commonly known to help combat diseases that are bacterial in nature. The antibacterial processes attributed are: an osmotic effect due to the content of sugar, the content of hydrogen peroxide (produced by the bee's added glucose oxidase), its acidity and other substances extracted from flowers. Honey, with its anti-H, has been researched *in vitro pylori* activity [31]. Manuka honey

comes from a single source of herb. This honey has been shown to have bacteriostatic properties at a 50 mL/L concentration against *H. pylori* [32]. 8 generic honey brands that were sold in Muscat, Oman were screened for anti-H in an *in vitro* assay. Operation of *pylori* by process of surface diffusion either in conjunction with amoxicillin or clarithromycin. The findings revealed that anti-H was present in both of them. *Pylori* activity, but no synergies with either honey and clarithromycin or honey and amoxicillin were observed [33]. These data show that a triple regimen with these honeys may allow the bacteria to be removed.

Green tea is very consumed drink and it can inhibit the growth of Green tea is very useful for the treatment of H. The existence of virus H. in vitro has been shown. These findings indicate that in vivo analysis of green tea administration is promising. Another research showed that green tea reduces the number of bacteria. C57BL/6J and pylori-infected C57BL/6J mice were given green tea prior to infection [33]. Another experiment was performed to assess the effect of green tea extract on the growth of H. pylori. it was shown that eradication of pylori contribute to higher antitubercular and antimicrobial levels. Antioxidants, the major antioxidant compounds in green tea also demonstrated antibacterial activity against Herpes Simplex virus. This is done in both in vitro and in vivo. Garcinia cambogia had the highest operation with a MIC value of 8 μ g/mL for 50% of the tested strains. In Mongolian gerbils with infections due to enteric bacteria, supplement of catechins in the diet (2 percent) for 2 weeks resulted in low rate of bacterial eradication (36 percent) and substantial decreases in blood loss and erosion [34]. The authors propose that combining catechins with proton pump inhibitors and delivery mechanisms that improves the gastric-transit time will increase the efficacy of the catechins. Green tea extract inhibits H. both of these drugs have an IC₅₀ value of 13 µg/mL Catechins were labeled as the active compounds, and hydroxyl group was noted as an essential site in the urease inhibition. We recognize that polyphenols found in green tea resist oxidative damages caused by hydrogen peroxide the VacA poison [35].

Glycyrrhiza glabra can be particularly effective for those suffering from peptic ulcers. Several studies have tested the antibiotic effect of liquor ice and some of its metabolites against Helicobacter pylori. Recently, a flavonoid rich extract of G. glabra was tested for anti-H1N1 activity to study of pylori and its action on bacteria The MIC of gut Gard was 32 - 64 μ g/mL and glabridin, the main flavonoid present in the extract, displayed a more potent action against

H. pyloric. The manner in which the drug functions is possible by inhibiting protein synthesis, DNA gyrase, or dihydrofolate reductase. However, the consent of *H. pylori* were not especially influenced by GANAP [36].

Garlic, Allium sativum, is a very useful food ingredient not only as an important flavouring factor in food but also for its medicinal properties. These symptoms are caused by garlic extract. There is considerable interest in researching garlic's protection and beneficial effects on H. There was a decline between the use of garlic as food and the incidences of stomach cancers [37]. In vitro and long clinical experience indicates pylori activity in extracts and compounds derived from garlic. Proof of other research have appeared, but not absolutely universally [38]. Aqueous garlic extract had a MIC value of approximately 40 µg/mL. The MIC value for other garlic compounds is 10 - 25 μg/mL [39]. However, in the few in vivo experiments that have been performed, the viability of the bacterium was not significantly compromised. The use of sliced garlic in the diets of patients didn't affect other metrics such as H's amount. pylori infection was confirmed by urease breath screening. Since taking garlic, blood markers rose and there were no advantages at all. When 4 percent of garlic powder was applied everyday to Mongolian gerbils for six weeks, similar effects were obtained. Pylori-induced gastritis, but in this situation, overall had reduced the presentation of the condition [40].

Red wine's antibacterial action against H. the bacteria has been checked. The scientific literature claims that a compound in the resveratrol family is, at least in part, the compound responsible for the action against the pathogen (MIC spectrum of 6.25 - 25 μg/ mL) [41]. The ultimate influence, however, was expressed as H [red wine] inhibits H [caffeine]. The urease activity increases. It is assumed that red wine can inhibit ion and urea conduction in cells, as well as cellular vacuolisation caused by VacA. These anti-inflammatory, anti-viral and anti-oxidant properties were related to its polyphenol properties; these compounds inhibit the VacA channel [42]. The findings suggest that many of the ingredients of drinking wines and green tea or a polyphenol combination (tannic acid and n-propyl gallate) given to humans enhance memory. Through injecting mice with pylori, or by injecting mice with VacA toxin, mice had less gastric damage but also had more damage in the colon from the bacteria. The finding that VacA inhibition can play a role in this novel protective effect is of concern. [43].

Vaccines

In those countries with a high incidence of infection, the need for a vaccine is highly evident; with increased susceptibility to the antibiotics used to treat it, which may increase the recurrence risk of the infection; and with high rates of morbidity and mortality caused by *H. pylori*-associated pathologies of infection [44]. High medical rates for diseases associated with this infection (i.e. gastric cancer and peptic ulcer) make the production of vaccination an option that is cost-effective. Therefore, it is important to provide appropriate vaccines that could eliminate and/or treat the infection or at least change the host-pathogen relationships in a way that inhibits disease progression.

Possible reasons of treatment delay

Resistance in strains of *Helicobacter pylori* antibiotics the risk factor for resistance to antibiotics used for the eradication of Helicobacter pylori may be diabetes. A better rate of H, whereas in a study in Taiwan, Pylori eradication has been observed in diabetes patients [45], several experiments have shown both positive and negative outcomes. The major causes of H. are reduced microvascular gastric ingestion reduction medications, gastroparesis and the use of antibiotics for chronic urogenital infections, respiratory infections and skin resistance. The resistance of pylori to routine care in diabetic patients. Approximately 40 per cent of patients with type 1 diabetes and 30 per cent of patients with type 2 diabetes are affected by diabetic gastroparesis, especially those with long-term Cosmin V. Obleaga., et al. 157 disease. In a thesis by Ojetti., et al. published H. pylori the rate of overgrowth of the bacteria associated with ulcers is lower in diabetics than in infants, possibly as a result of more regular invasions and antibiotic therapies. The Bismuth-based treatment is more effective in treating *H. pylori*. It is possible that eradicating these diseases would boost the situation dramatically [45]. When a patient is treated for recurrent respiratory infections, they often are treated with antibiotics, and the bacteria will become resistant to the drugs used even within different regimens for *H. pylori* eradication scheme, amoxicillin, clarithromycin, metronidazole and tetracycline are antibiotics used in first-line treatment of various respiratory or urogenital tract infections; in many cases, patients undergoing treatment for H. pylori infection will require treatment with antibiotics. These antibiotics have been used for treating other diseases, such as, gonorrhea and cholesterol. This pattern is disturbing because it rejects the use of scientific procedure without fair experimentation first [46]. While in the same research conducted by Megraud F in the American Journal of Medicine, an important positive link was identified between the use of antibiotics in ambulatory treatment and in the primary degree of resistance demonstrated in antimicrobial main agents used to remove H. pylori. Awareness about the antibiotic drugs used in a certain area or by every patients can be provide useful information about the antibiotic susceptibility or tolerance of H. pylori to various antibiotics. H. pylori strains that develop in the presence of cholesterol are more resistant to prolonged exposure to several antibiotics. It is understood that such kind of antibiotics and cholesterol increase into the blood, because of this kind of resistant bacteria that live on our bodies and are infecting us pyloric co infections, which are triggering "recent" work to show that. Bile salts allow certain bacteria to become more susceptible to antibiotics [47]. This suggests that H. pylori can use the cholesterol modifying its envelope so as to resist to multiple antibiotics. Before using this antibiotic, it is necessary to note that it is an antibiotic that prevents protein synthesis. Infection with the bacteria Helicobacter pylori. A research was performed on the resolution of these bacteria as they are exposed to antibiotics. Human H. pylori, grown in the presence or absence of cholesterol, demonstrated how cholesterol significantly increased H density. Some bacteria have been immune to the tetracycline and clarithromycin. Both antibiotics hinder the DNA replication, such as Cipro and metronidazole and they are also used for the treatment of H(ATSA). People can suffer from gastritis and gastric ulcers. Even though, H. pylori containing cholesterol that was grown with ciprofloxacin was more resistant to seizure. The bacteria grown without cholesterol. H. Using cultured pylori from patients with a cholesterol mutation, scientists have found that H. pylori are immune to certain antibiotics by using these mutations (about 10 to 30 times). Like Cox-2, in the case of antibiotics that prevent the biosynthesis the cell membrane, i.e., ampicillin and amoxicillin, there were also related results: H. When grown on a medium with cholesterol levels, bacteria strains developed up to 1,000 times more resistant to antibiotics than those which were grown without cholesterol. Bismuth is part of the routine of her treatment of HIV. P. urealyticum infections in some countries. H. pylori evolved more effectively with cholesterol as a food than with a nutrient that was not cholesterol-rich. Pyloris bacteria are grown without cholesterol. H. it is observed when pylori (digestive bug) are grown without cholesterol that they are often more prone to rifampicin than H The bacteria cultured with cholesterol [48].

Conclusion

It is also a very major issue with *Helicobacter pylori* infection, even experts have difficulty with the diagnosis and the treatment. Prophylaxis of such disorders such as peptic ulcer, gastric lymphoma of mucosa associated lymphoid tissue (MALT) and gastric cancer is the prevention, the effective therapy and the revaluation of its efficacy. These diseases are life-threatening for the patient and their severe complications (bleeding, perforation). Treatment failure is due to the non-cooperation of the patient or his/her antibiotic resistance; it depends with such cases depending on the country of origin of the patient, the patient him/herself, and past antibiotic prescriptions. If the second procedure (as suggested by regional doctors) still fails, it is important to get an endoscopy in order to produce a biopsy sample from which the culture and DST can be performed. Even though H. pylori. In about 20 per cent of cases, pylori treatment fails, moral support for the patient by the clinician, information on possible evolutionary complications of H. pylori infection and the patient's periodic evaluation during treatment are important instruments on which therapeutic success depends.

Bibliography

- Marshall BJ and Warren JR. "Unidentified curved bacilli in the stomach of patients with gastritis and peptic ulceration". *Lan*cet 1 (1984): 1311-1315.
- Amieva MR and El-Omar EM. "Host-bacterial interactions in Helicobacter pylori infection". Gastroenterology 134 (2008): 306-323.
- Peura DA and Crowe CE. "Helicobacter pylori". In: Feldman M FL, Brandt LJ, editors. Feldman: Sleisenger and Fordtran's Gastrointestinal and Liver Disease. 9th edition. Philadelphia: Saunders (2010): 833-845.
- Fock KM and Ang TL. "Epidemiology of Helicobacter pylori infection and gastric cancer in Asia". European Journal of Gastroenterology and Hepatology 25 (2010): 479-486.
- Infection with Helicobacter pylori. In: IARC monographs on the evaluation of the carcinogenic risks to humans. Schistosomes, liver flukes and Helicobacter pylori". Lyon, France: International Agency for Research on Cancer 61 (1994): 177-240.

- Malaty HM and Graham DY. "Importance of childhood socioeconomic status on the current prevalence of *Helicobacter pylori* infection". *Gut* 35 (1994): 742-745.
- 7. Rowland M., *et al.* "Low rates of *Helicobacter pylori* reinfection in children". *Gastroenterology* 117 (1999): 336-341.
- 8. Parsonnet J., et al. "Fecal and oral shedding of *Helicobacter pylori* from healthy infected adults". *The Journal of the American Medical Association* 282 (1999): 2240.
- 9. Tindberg Y., et al. "Clinical symptoms and social factors in a cohort of children spontaneously clearing *Helicobacter pylori* infection". *Acta Paediatrica* 88 (1999): 631-635.
- Rothenbacher D., et al. "Prevalence and determinants of Helicobacter pylori infection in preschool children: A population based study from Germany". International Journal of Epidemiology 27 (1998): 135-141.
- 11. Gold BJ. "Helicobacter pylori infection in children". Current Problems in Pediatric 31 (2001): 247-266.
- Rocha GA., et al. "Transmission of Helicobacter pylori infection in families of preschool-aged children from Minas Gerais, Brazil". Tropical Medicine and International Health 8 (2003): 987-991.
- Malaty HM., et al. "Helicobacter pylori infection in asymptomatic children: Impact of epidemiological factors on accuracy of diagnostic tests". Journal of Pediatric Gastroenterology and Nutrition 35 (2002): 59-63.
- 14. Ndip RN., et al. "Helicobacter pylori antigens in faeces of asymptomatic children in the Buea and Limbe health districts of Cameroon: A pilot study". Tropical Medicine and International Health 9 (2004): 1036-1040.
- 15. McCallion WA., et al. "Helicobacter pylori in children: Relation with current household living conditions". Gut 39 (1996): 18-21.
- Jones NL., et al. "Joint ESPGHAN/NASPGHAN Guidelines for the Management of Helicobacter pylori in Children and Adolescents (Update 2016)". Journal of Pediatric Gastroenterology and Nutrition 6 (2017): 991-1003.
- 17. Oderda G., *et al.* "Detection of *Helicobacter pylori* in stool specimens by non-invasive antigen enzyme immunoassay in chil-

- dren: multicentre Italian study". *British Medical Journal* 320 (2000): 347-348.
- 18. Jones DM., *et al.* "Campylobacter like organisms on the gastric mucosa: culture, histological, and serological studies". *Journal of Clinical Pathology* 37 (1984): 1002-1006.
- 19. Marshall BJ., et al. "Pyloric campylobacter serology". Lancet 2 (1984): 281.
- 20. Warren JR and B Marshall. "Unidentified curved bacilli on gastric epithelium in active chronic gastritis". *Lancet* I (1983): 1273-1275.
- 21. Furuta T and Graham DY. "Pharmacologic Aspects of Eradication Therapy for *Helicobacter pylori* Infection". *Gastroenterology Clinics of North America* 39.3 (2010): 465-480.
- Sugimoto M and Furuta T. "Efficacy of tailored Helicobacter pylori eradication therapy based on antibiotic susceptibility and CYP2C19 genotype". World Journal of Gastroenterology 20.21 (2014): 6400-6411.
- 23. Hori Y., et al. "1-[5-(2-Fluorophenyl)-1-(pyridin-3-ylsulfonyl)-1Hpyrrol-3-yl]-N-methylmethanamin e monofumarate (TAK438), a novel and potent potassium-competitive acid blocker for the treatment of acid-related diseases". *Journal of Pharmacology and Experimental Therapeutics* 335.1 (2010): 231-238.
- 24. Trespalacios AA., *et al.* "Impacto de la resistencia de *Helico-bacter pylori* a los antimicrobianos en la eficacia de la terapia triple estándar y en dos triples terapias con levofloxacina en pacientes colombianos". *Gastroenterol Latinoam* 23 (2012): S35.
- 25. Graham DY and Dore MP. "Helicobacter pylori therapy: a paradigm shift". Expert Review of Anti-infective Therapy 14.6 (2016): 577-585.
- 26. Trespalacios-Rangél AA., *et al.* "Surveillance of Levofloxacin Resistance in *Helicobacter pylori* Isolates in Bogotá-Colombia (2009-2014)". *PLoS One* 11.7 (2016): e0160007.
- 27. Crabol Y., *et al.* "Rifabutin: where do we stand in 2016?" *Journal of Antimicrobial Chemotherapy* 71.7 (2016): 1759-1771.

- 28. Gisbert JP and Calvet X. "Review article: rifabutin in the treatment of refractory *Helicobacter pylori* infection". *Alimentary Pharmacology and Therapeutics* 35 (2012): 209-221.
- 29. Lu H., et al. "Bismuth containing quadruple therapy for Helicobacter pylori: lessons from China". European Journal of Gastroenterology and Hepatology 25 (2013): 1134-1140.
- 30. Mohammadi M., et al. "Furazolidone, an Underutilized Drug for *H. pylori* Eradication: Lessons from Iran". *Digestive Diseases and Sciences* 62.8 (2017): 1890-1896.
- 31. Ali AT., et al. "Inhibitory effect of natural honey on *Helicobacter* pylori". *Tropical Gastroenterology* 12 (1991): 139-143.
- 32. Somal N., *et al.* "Susceptibility of *Helicobacter pylori* to the antibacterial activity of manuka honey". *Journal of the Royal Society of Medicine* 87 (1994): 9-12.
- 33. Stoicov C., et al. "Green tea inhibits *Helicobacter* growth in vivo and in vitro". *International Journal of Antimicrobial Agents* 33 (2009): 473-478.
- 34. Mabe K., et al. "In vitro and in vivo activities of tea catechins against *Helicobacter pylori*". *Antimicrobial Agents and Chemotherapy* 43 (1999): 1788-1791.
- 35. Tombola F, *et al.* "Plant polyphenols inhibit VacA, a toxin secreted by the gastric pathogen *Helicobacter pylori*". *FEBS Letters* 543 (2003): 184-189.
- 36. Krausse R., et al. "In vitro anti-Helicobacter pylori activity of Extractum liquiritiae, glycyrrhizin and its metabolites". Journal of Antimicrobial Chemotherapy 54 (2004): 243-246.
- 37. Steinmetz KA and Potter JD. "Vegetables, fruit, and cancer prevention: a review". *Journal of the American Dietetic Association* 96 (1996): 1027-1039.
- 38. Cellini L., et al. "Inhibition of Helicobacter pylori by garlic extract (Allium sativum)". FEMS Immunology and Medical Microbiology 13 (1996): 273-277.
- 39. Ohta R., et al. "In vitro inhibition of the growth of Helicobacter pylori by oil-macerated garlic constituents". Antimicrobial Agents and Chemotherapy 43 (1999): 1811-1812.
- 40. Iimuro M., *et al.* "Suppressive effects of garlic extract on *Helicobacter pylori*-induced gastritis in Mongolian gerbils". *Cancer Letters* 187 (2002): 61-68.

- 41. Mahady GB., et al. "Resveratrol and red wine extracts inhibit the growth of CagA+ strains of *Helicobacter pylori* in vitro". *The American Journal of Gastroenterology* 98 (2003): 1440-1441.
- 42. Tombola F., *et al.* "Plant polyphenols inhibit VacA, a toxin secreted by the gastric pathogen *Helicobacter pylori*". *FEBS Letters* 543 (2003): 184-189.
- 43. Ruggiero P., et al. "Red wine and green tea reduce H pylorior VacA-induced gastritis in a mouse model". World Journal of Gastroenterology 13 (2007): 349-354.
- 44. Graham DY and Fischbach L. "Helicobacter pylori treatment in the era of increasing antibiotic resistance". Gut 59 (2010): 1143-1153.
- 45. Tseng CH. "Diabetes, insulin use and *Helicobacter pylori* eradication: a retrospective cohort study". *BMC Gastroenterology* 12 (2012): 46.
- 46. Demir M., et al. "Bismuth-based first-line therapy for *Helico-bacter pylori* eradication in Type 2 diabetes mellitus patients". *Digestion* 82.1 (2010): 47-53.
- 47. Simrén M., *et al.* "Intestinal microbiota in functional bowel disorders: a Rome foundation report". *Gut* 62.1 (2013): 159-176.
- 48. McGee DJ., *et al.* "Cholesterol Enhances *Helicobacter pylori* Resistance to Antibiotics and LL". *Antimicrobial Agents and Chemotherapy* 55.6 (2011): 2897-2904.

Assets from publication with us

- $\bullet \quad \hbox{Prompt Acknowledgement after receiving the article}$
- Thorough Double blinded peer review
- Rapid Publication
- Issue of Publication Certificate
- · High visibility of your Published work

Website: www.actascientific.com/

Submit Article: www.actascientific.com/submission.php

Email us: editor@actascientific.com

Contact us: +91 9182824667