Molecular detection of *Helicobacter pylori* among gastroduodenitis and peptic ulcer patients in Khartoum state

Abbas Bakhit Mohammed Rahama
Al-Neelain University, Sudan

*Helicobacter pylori* infection is associated with gastroduodenitis, gastric ulcer and duodenal ulcer. Many of studies have released causes of gastric ulcer and duodenal ulcer (approximately 95% of duodenal ulcers and 85% of gastric ulcers) to infection with *H. pylori*. *Helicobacter pylori* (*H. pylori*), previously named *Campylobacter pyloridis*, is a Gram-negative, microaerophilic bacterium found in the stomach. *H. pylori*, a spirally shaped bacterium, 0.5-0.9 μm wide by 2-4 μm long. Like *Campylobacter*, it requires carbon dioxide for growth, but it has a tuft of sheathed unipolar flagella, unlike the unsheathed flagella of *Campylobacter*. *H. pylori* was identified in 1982 by Barry Marshall and Robin Warren, who found that it was present in patients with chronic gastritis and gastric ulcers, conditions that were not previously believed to have a microbial cause. It is also linked to the development of duodenal ulcers and stomach cancer. However, over 80% of individuals infected with the bacterium are asymptomatic and it has been postulated that it may play an important role in the natural stomach ecology. In 1893, the Italian researcher Giulio Bizzozero described helical shaped bacteria living in the acidic environment of the stomach of dogs. *Helicobacter pylori*, is the main cause of chronic active gastritis and has major role in development of duodenal ulcer, also associated with but not necessary the cause of gastric carcinoma ASM. There is 80% of chronic gastritis caused by *H. pylori*. Since the discovery of *Helicobacter pylori* in the 1989 by Warren and Marshal, much has been learned about these Gram-negative spiral bacteria and its associated disease states. The study was aimed to detect *Helicobacter pylori* in patients with gastroduodenitis and peptic ulcer in Khartoum state by employing Polymerase Chain Reaction (PCR) to detect *H. pylori* 16S gene, Sudan. Molecular testing for *H. pylori* 16-S gene was done on 57 stomach and duodenal biopsy specimens using PCR technique. Biopsy specimens were collected by gastroenterologist using endoscopy. Multiple gastric biopsy specimens were taken from the stomach antrum and the corpus and the duodenum. Transport of specimens was in normal saline and kept at -80 °C till used. Extraction was done by using Vivantis GF-1 Nucleic acid extraction kit (Vivantis, Malaysia). The amplification reaction was carried out in thermo cycler machine PCR system with program system consisting of (1 min at 94 °C, 2 min at 55 °C, 3 min at 72 °C) and final extension was done at 72 °C for 5 min) PCR products were separated in a 1.5% agarose gel, then stained with ethidium bromide and viewed under gel documentation system. A result was considered positive when a band of the appropriate size was visible in the gel. Standard procedures for reducing contamination were strictly followed. Twelve samples (21.1%) out of 57 were positive by PCR, while 45 samples (78.9%) were negative. In conclusion the frequency of 16 S rDNA genes of *H. pylori* among endoscopic patients was 21.1%.

Effect of Ofloxacin autogel on periodontopathic bacteria in chronic periodontitis

Abeer S Gawish¹,²
¹King Abdulaziz University, KSA
²Al-Azhar University, Egypt

A n appreciated treatment modality for periodontitis is scaling and root planning followed by a controlled-release antimicrobial intra-pocket delivery system. The aim of the present work was to develop a smart controlled-release liposomal autogel system of ofloxacin and evaluate it for the management of aggressive periodontitis in adult patients. Autogels based on chitosan neutralized by β-glycerophosphate was prepared after setting a high degree of deacetylation for chitosan. The systems were characterized for muco-adhesion, syringeability and gelation onset. In vitro ofloxacin release in pH6.8 revealed about 83% release after 3 days. Further release modulation was done by encapsulation of ofloxacin in liposomes. Upon inclusion into the gel, liposomes afforded 80% of drug release in 7 days. When tested microbiologically in adult volunteers the ofloxacin liposomal autogel demonstrated markedly lower anaerobes bio-burden in sub-gingival plaque samples than ofloxacin solution after 7days. Moreover, the liposomal autogel formula showed significant improvement in the different clinical parameters evaluated. It can be concluded that the developed ofloxacin liposomal autogel is promising in the management of aggressive periodontitis in adults.