Introduction

Ulcerative Colitis (UC) is an inflammatory bowel disease (IBD) of unknown etiology that is confined to the large bowel mucosa. It seems to be a multifactorial disorder involving both genetic and environmental components, particularly the bacterial gut microbiota (Adiana et al., 2004).

The etiopathogenesis of IBD is poorly understood; however, two general hypotheses exist. First, the disease is due to an abnormal and uncontrolled immune response to luminal antigen, and second the disease is initiated by an appropriate immune response to an anterior pathogen, which is still unidentified (Rachel et al., 1997).

*Helicobacter* species are divided into two subgroups. The better known gastric *Helicobacter* species, which preferably colonize the host’s stomach, represent only one-third of the known species of Helicobacteraceae. The remaining two third of *Helicobacter* species are referred to as enterohepatic because they predominantly colonize the intestine and the hepatobiliary system (Ulrich et al., 2004).

Recently, enterohepatic *Helicobacter* species have been discovered in inflammatory bowel disease (IBD) of rodents, carnivores, and primates. Although to date most research has focused on Helicobacter pylori infection in humans, evidence from animal studies and immunocompromised patients suggests that, other
*Helicobacter* species exist in the large bowel. Several nongastric *Helicobacter* species have been reported to infect immunocompetent and immunocompromised humans, infection has been associated with gastroenteritis and in the case of two species, *Helicobacter cinaedi* and *Helicobacter fennelliae*; human infection has been associated with colitis and proctitis (*Rachel et al., 1997*).