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Need To Act Hastily against the Gastric Cancer Pathogen *Helicobacter Pylori*

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Introduction

Helicobacter pylori is one of the most prevalent gastric pathogens and colonizes 50% of the global population. Robin Warren and Barry J. Marshall discovered the gastric pathogen *H. pylori* from gastric biopsy samples and their discovery was awarded the Nobel Prize in Physiology or Medicine in 2005. *H. pylori* colonize the stomach and can cause the gastric mucosal inflammatory process termed "gastritis." The discovery of H. pylori challenged the prevailing view concerning peptic ulcer etiology. *H. pylori is* the only bacterium known for its common cause of bacterial-induced cancer. As a result, understanding the association of *H. pylori* with gastric cancer is a significant issue warranting further studies [1]. It is no wonder the bacterium was classified as a Class II carcinogen, since the risk ratio of gastric cancer with *H. pylori* infection was 5.8% compared to the healthy stomach individual [2] and high prevalence of *H. pylori* association reporting in East Asia [3]. The reason why the bacterium induces gastric cancer is due to the oncogenic protein Cag A synthesized and injected to host epithelium by the type IV secretion system, which plays a major role in *H. pylori* induced gastric cancer [4]. Notably, *H. pylori* eradication decreased the risk of developing gastric cancer in *H. pylori*-infected patients [5]. Bismuth quadruple therapy has been shown to be a successful eradication therapy in patients in the clarithromycin resistance geographical region or patients on a 14-day course of triple therapy which typically contain two antibiotics such as clarithromycin and amoxicillin with one proton pump inhibitor (PPI) [6] considering a successful medication for *H. pylori*. Individuals from low socio-economic backgrounds are more prone to developing this disease and are also highly prone for disease recurrence after an eradication therapy [7]. The world is facing an emerging serious and potent threat as a result of antibiotic resistance and *H. pylori* (clarithromycin resistant *H. pylori*) is one of the culprits on the list of serious threats announced by the World Health Organization (WHO) and Center for Disease Control (CDC) [8]. The pretreatment resistance is also an emerging problem in managing *H. pylori* and its treatment [9]. The point mutation occurred in the 23s rRNA gene drives clarithromycin resistance [10]. One of the main reason for increasing resistance to clarithromycin is due to the consumption of macrolides antibiotics previously and the occurrence of clarithromycin resistance in children is also due to intake of uncontrolled macrolides for respiratory tract illness [11]. Hence, the development of novel drugs targeting *H. pylori* and its drug resistant strains, with anti-bacterial and anti-cancer activity for the treatment of *H. pylori* and its role in gastric cancer is imperative. High-throughput screening (HTS) is a valuable and cost-effective process to discover novel antimicrobials [12] and very few HTS studies are reported to discover molecules against *H. pylori*. One such pilot-HTS study reported that the anthelmintic molecule niclosamide can be repurposed against *H. pylori* [13]. HTS can be an effective component of studies to discover novel and potential molecules to combat *H. pylori* infection, *H. pylori* drug resistance, and *H. pylori* infection that leads to gastric cancer given the risks posed by antibiotic resistance.

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