Short Communication

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Helicobacter Pylori and Antibiotic Resistance

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ABSTRACT

H. pylori (*H. pylori*) is a global health problem and it is important to inform the general population with the aspects of this infection. *Helicobacter pylori* eradication is becoming increasingly difficult. High prevalence of *H. pylori* infection, the complexity of its treatment, poor patient adherence to the treatment may contribute to antibiotic resistance and healthcare costs.

Keywords: *Helicobacter Pylori*; Antibiotic Resistance

Abbreviations: MALT Lymphoma: Extranodal Marginal Zone Lymphoma of Mucosa-Associated Lymphoid Tissue; 23S rRNA: Component of the Large Subunit (50S) of the Bacterial/Archean Ribosome

Introduction

Helicobacter pylori (H. pylori) has been established as the most prevalent chronic infection globally, that, has affected more than a half of the world population [1]. *Helicobacter pylori* is a Gram-negative bacterium that has been classified within the *Epsilonproteobacteria* class, under the order *Campylobacterales*, within the *Helicobacteraceae* family, and is part of the *Helicobacter* genus. To date, more than 40 species within the Helicobacter genus have been identified and categorized, capable of colonizing the stomach (e.g., *H. pylori*) or intestines (e.g., *H. cinaedi and H. fennelliae*) [2]. Humans are the primary hosts for *H. pylori*, with evidence suggesting that the bacterium is transmitted through oral or fecal-oral pathways [3,4]. This bacterium predominantly colonizes the gastric mucosa, though it has also been detected in dental plaque and saliva of infected individuals. The International Agency for Research on Cancer and the World Health Organization (WHO) have also classified *H. pylori* as a first category carcinogen, due to its significant role in the etiology of stomach cancer, highlighting the potential for cancer prevention through early eradication of the bacterium [5]. Infection with *H. pylori* poses a substantial clinical challenge, as it is associated with conditions such as gastritis, gastric and duodenal ulcers, MALT lymphoma, and stomach cancer [6-9].

Importantly, treating *H. pylori* infection in populations at high risk has been shown to decrease the incidence of stomach cancer among asymptomatic individuals [10]. The standard treatment regimen for *H. pylori* infection combines antibiotics, antisecretory agents, and proton pump inhibitors [11]. Initially, therapy often involves a combination of clarithromycin, amoxicillin, metronidazole, and proton pump inhibitors, with clarithromycin serving as a primary treatment option due to its effectiveness against the infection [12]. Should initial treatment fail, secondary therapies, such as triple therapy with levofloxacin (comprising levofloxacin, amoxicillin, and a proton pump inhibitor), are considered [13]. However, the global increase in *H. pylori* resistance to antibiotics presents a significant challenge [14]. Resistance rates exceed 15% for clarithromycin, range between 45% to 55% for metronidazole, and between 14% to 20% for levofloxacin [15]. Resistance to clarithromycin and levofloxacin primarily arises from point mutations in the bacterium's genetic material, which alter the antibiotics' target sites and interfere with drug activity [16]. In Croatia, a rise in primary resistance to these antibiotics in *H. pylori* has been noted, with mutations identified in the 23S rRNA, gyrA, and gyrB genes affecting clarithromycin and levofloxacin resistance by modifying target sites or protein structures, thereby diminishing treatment efficacy [17].

Conclusion

Molecular docking analyses have shown that *H. pylori* strains harboring resistance-related mutations exhibit reduced susceptibility to clarithromycin and levofloxacin compared to wild-type strains, due to altered non-covalent interactions (e.g., hydrogen bonds, ionic interactions) that weaken antibiotic-protein binding, leading to antibiotic resistance. The occurrence of dual resistance to clarithromycin and levofloxacin highlights the bacterium's evolving resistance to different antimicrobials, posing an increased health risk. Research of Samanic et al. into *H. pylori*'s antibiotic resistance represents a crucial step towards a more comprehensive understanding of this issue, suggesting that a broader research approach could shed light on the intricate interplay between patient characteristics, *H. pylori* genetics, and antibiotic resistance at a molecular level [17]. Further studies, including molecular dynamics, could reveal the dynamic nature of antibiotic-target site interactions in the bacterium, offering insights into antibiotic resistance mechanisms at an atomic level. Such in-depth research is vital for advancing *H. pylori* treatment strategies, contributing to the development of targeted therapies, and addressing the challenge of antibiotic-resistant strains.

Conflict of Interest

Authors have no conflict of interests associated with this article.

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