

Mini Review

## Helicobacter pylori Infection: Protective Agent against Mycobacterium tuberculosis

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### Introduction

Tuberculosis (TB) is one of the world's most deadly and contagious diseases (eight and two millions of new infected cases and deaths annually, respectively) [1]. Similarly to other infectious agents, antibiotic therapies are applied to eliminate this microorganism in symptomatic individuals. Strikingly, due to the frequently failed therapies, we have observed increasing numbers of resistant microorganisms in the biologic world [2]. Given the insertions, polymerase errors, duplications and deletions in bacterial chromosomes, all prokaryotes proliferate with a considerable rate of mutations [3]. Collectively, abovementioned mechanisms provide a wide set of possible mutations for Mycobacterium tuberculosis (M. tuberculosis). To date, several mutations were discovered as main causes of antibiotic resistance in M. tuberculosis, but other contradictory results are also suggest other mutation sites as well [1,2]. Altogether, because of particular biologic features of TB bacilli, the genetic basis of antibiotic resistance for most anti-tuberculosis drugs is not clearly known. Apart from its ability to become resistant against most of the prescribed antibiotics, the bacteria seem to be able to overcome immune response and escape from effective cellular and humoral immunity. In other words, TB chronic infection seems to be sustained by a complex and mostly unknown interplay of human immune responses, which increases the residence time of this persistent bacterium in pulmonary cavities. The success of M. tuberculosis as one of the most prevalent bacterial pathogens is widely affected by its ability to survive in respiratory cells,

where antibiotics cannot reach the optimal concentration to achieve bactericidal effects. To date, an exact mechanism of interplay between the M. tuberculosis and the host, which end in chronic or latent infection, is largely under debate. For a long time, antibiotic resistance and tuberculosis patients were defining as major problem in health. In recent years, even following application of new antibiotics (natural and synthesized), successful eradication of this bacilli have not been achieved. Accordingly, TB patients can still carry M. tuberculosis in their clinical samples after antibiotic therapies, thus we need to consider better intervention to defeat the bacterium in positive cases. Antibiotic resistant tuberculosis poses a major threat to human health and calls for urgent intervention. The chemotherapy for tuberculosis is relatively challenging compared to other bacterial infections. Long generation time, and also the ability to become latent, has provided difficulties in applying the usual antibiotic therapy against this bacterium. Following many failed antibiotic therapies against M. tuberculosis, we aim to provide a novel therapeutic and preventive approach to battle against M. tuberculosis.

**Keywords:** H. pylori; Immune System; M. tuberculosis; Treatment

### H. pylori infection against M. tuberculosis: (Novel approach)

The human immune system clears many of the infectious

agents that we face during our lifetime; however some pathogens can stay in our body as persistent or latent agents. Currently, more than 35% of the world's population is latently infected with the *M. tuberculosis* [4]. Moreover, *Helicobacter pylori* (*H. pylori*), which colonizes more than 60% of the world population, is the most important cause of chronic gastritis, and duodenal ulcers, and likely of gastric ulcers [5]. Taken together, it has been asserted that the *H. pylori* and *M. tuberculosis* are the most prevalent bacterial pathogens that have ever been investigated [6]. Recent findings showed that *H. pylori* infection can prevent colonization by *M. tuberculosis* in the human respiratory system [7-9]. Certainly, *H. pylori* is a very diverse microorganism, which infects half of the world population. Given the fact that *H. pylori* is genetically variable, it is likely possible to identify a harmless strain and test its ability to induce an effective immune response against *M. tuberculosis*. Of note, there is a complex immune response in order to eradicate reactive TB infection [7]; a fact that brings an idea about *H. pylori* colonization due to the amazing ability to adjust human immune system. Broadly defined, certain human microbiota can affect pathogenic colonization [10]. Basically, it can be hypothesized that *H. pylori* can provoke immune response in order to suppress TB infection. Currently, it has been generally indicated that an IFN- $\gamma$  driven Th1-type response is undoubtedly necessary for maintenance of TB latency [11]. In other words, *H. pylori* infection was associated with an enhanced IFN- $\gamma$  and other Th1-type cytokine in responses to TB infection. Furthermore, treatment of TB is mostly accompanied with severe side effects such as hepatitis, fever, itching, and polyneuropathy [12]. The critical matter of those severe side effects provokes an idea to avoid antibiotic therapies against *M. tuberculosis*. Notably, new investigations are required to elucidate the mechanism of contribution of *H. pylori* infection as a protective immune response to TB infection. Conclusively, *in vitro* and *in vivo* tests can provide us more detailed information about current proposed strategy to deal with this mysterious bacterium, which stays with humans over hundreds of years. Since current adopted strategies including antibiotic therapies have failed to eliminate *M. tuberculosis*, application of this new proposed strategy to use harmless and non-virulent *H. pylori* strains might be the missing key to eradicate *M. tuberculosis*.

### Conflict of interest

The author declares that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

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