Letter to the Editor

Comment on “Helicobacter pylori Outer Membrane Protein 18 (Hp1125) Is Involved in Persistent Colonization by Evading Interferon-γ Signaling”

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We read with interest the paper by Shan et al. [1] in a recent issue. It is an interesting paper concluding that Helicobacter pylori (H. pylori) omp18 is indirectly affecting long term bacterial colonization by successfully influencing IFN-γ-mediated immune response. Nevertheless, we found that some statements could not support the final conclusion. H. pylori infects the gastric mucosal layer of half of the human population worldwide and causes various digestive disorders such as chronic gastritis, gastric ulcer, duodenal ulcer, and gastric cancer [2]. To date, it has been established that such complex mechanism of bacterial interaction with human host can shape the successful and persistent colonization of H. pylori [3,4]. Undoubtedly, understanding the mechanisms of immune evasion could provide new options for better management of infection. To our knowledge, the host immune response to the infection is ineffective; accordingly, the bacterium persists and remains for decades. In brief, Shan et al. [1] reported the oipA as a critical factor affecting bacterial colonization. However, we know that, in chronic process of colonization adopted by H. pylori, the connection of a unique factor to the drive of the final pattern of this phenomenon could be too speculative. Despite the interesting report of Shan et al. [1], we may hypothesize more factors involved in H. pylori colonization. Surprisingly, H. pylori colonization is not comparable with that of other pathogens [5]. Indeed, different mechanisms are contributing to this mysterious and long term biologic function. Conclusively, more studies are necessary to draw a direct and final conclusion on “the mystery” of H. pylori colonization.

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Conflict of Interests

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References


