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Editorial
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How Does the Immune Response Influence the Risk of Developing Gastric Cancer in Different Age Groups?

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Editorial

Helicobacter pylori are a highly studied bacterium that inhabits the gastric environment of more than half the world's population. The infection is acquired during childhood and persists for the rest of life if left untreated. It is not new that this infection predisposes to the development of some gastric diseases during adulthood such as gastric carcinoma [1]. Although this infection is a preponderant factor in the development of gastric cancer, it is important to be highlighted that other factors, like host characteristics, are also important. With regard to the peculiarities of the host, the immune system plays a crucial role in the triggering and evolution of gastric carcinogenesis. Host cytokines as IL-1β, IL-8 and TNF-α seems to be crucial in the immune response related to the infection. Furthermore, some of them are directly involved in the development of specific gastric lesions and tumorigenesis [2].

The gastritis caused by *H. pylori* infection in childhood tends to be milder than adulthood. In fact, a study found that higher concentrations of regulatory T cells (Treg) and a significant increase of IL-10 and TGF-β1 were observed in *H. pylori*-positive children mucosal layer in comparison with infected adults [3]. This phenomenon provides a regulation of gastric inflammation, what might reduce the risk of gastric lesions development and, consequently, gastric cancer. However, this cytokine profile also avoids an effective immune response against the pathogen, preventing its elimination. Besides that, the immune response triggered in children gastric environment seems to be important in the development of gastric malignancy during adulthood. In a study conducted in a Brazilian population, the predominance of Treg in the gastric mucosa of *H. pylori*-positive children was also observed. In addition, this study demonstrated an exuberant expression of IL-17 in the gastric environment of infected adults [4]. This fact might contribute to an inflammatory response that likely adds to the development of gastric lesions, which can lead to gastric carcinogenesis.

Interestingly, a study that compared healthy individuals with gastric cancer patients found that there is a significant increase in Th17 cells in the last group. Furthermore, the Th17 cells count was directly associated with the clinical stage of the disease and a substantial increase of Th17 cells was observed in lymph nodes with tumor drainage and in advanced disease [5]. Although this study did not consider *H. pylori* status, these results contribute to ratify the importance of IL-17 role on gastric carcinogenesis.

The levels of cytokines released by Th1 cells also seems to vary between the different age groups of *H. pylori*-positive patients, being observed higher concentrations of these molecules in adults when compared to children in a Brazilian population. The Th1 inflammatory response in adults have been associated with gastritis, that, along with some host predispositions and environmental factors, can lead to gastric atrophy, metaplasia and other precancerous lesions. Raised gastric concentrations of IFN-γ were observed in patients aged 19 to 39 years when compared to individuals aged 14 to 18 years. However, the levels of this cytokine, which have an important effect against tumorigenesis, tend to decrease in older patients, who are at higher risk of carcinogenesis [6].

In conclusion, the variations between the different profiles of the immune response in distinct age groups are determinant in the infection outcomes. Assuming that *H. pylori* colonization occur in early life and that the immune response patterns vary over time, more studies on how do these different immune modulations reverberate on neoplastic processes are necessary to enable a better understanding about gastric carcinogenesis.

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