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Physiology and Molecular Changes in Gastric Infection by H. Pylori: Mini-Review

Prudente RCS[1], Santos GR[1,2], Spricido L[1,2], Jorgino FR[3], Bengami MD[1,4], PINTO CAL[1,4]

[1]*Department of Morphology and Pathology, Jundiaí Medical School, Brazil*

[2]*Department of Life Sciences, Paulist University (UNIP), Brazil*

[3]*Department of Surgery, Jundiaí Medical School, Brazil*

[4]*Department of Anatomic Pathology, AC Camargo Cancer Center, Brazil*

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***Corresponding author:** Pinto Cal, Department of Anatomic Pathology, AC Camargo Cancer Center, São Paulo city, State of São Paulo, Brazil
Email: patologia@fmj.br

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Abbreviations: LPS: Lipopolysacharides; VaC A: Vacuolization Cytoxin A; CagA: Cytotoxin Associated Gene A; MAP: Microtubules Associated Protein

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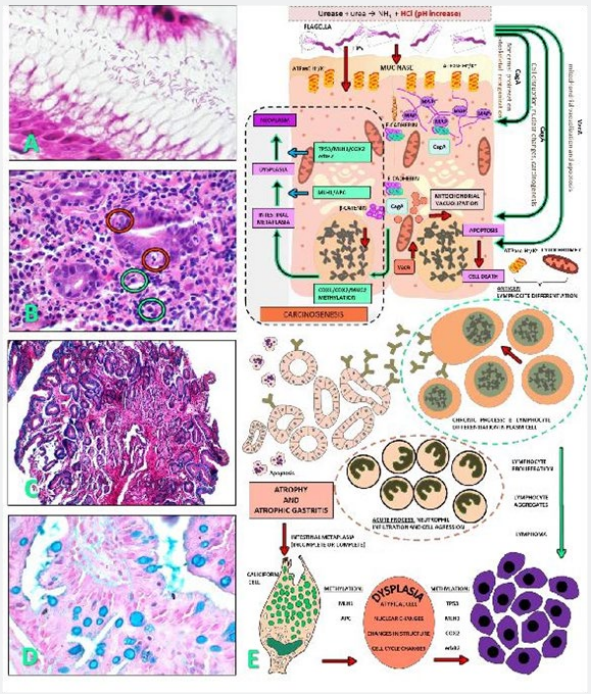

Figure 1:

A. H. pylori
B. Glandular aggression.
C. Mucosa atrophy
D. Intestinal metaplasia.
E. Schematic picture of the infection by H. pylori bacterium.

Helicobacter pylori (H. pylori) is a gram negative, spiral and flagellated bacterium, being main etiologic agent responsible by physiologic gastric disorders as chronic gastritis. This bacterium has several factors of virulence that causes damages in gastric cells, being able take to development of neoplasm. In this review let´s discuss the main virulent factors of the H. pylori and the physiologic repercussions in organism. H. pylori is one of few microorganism that colonize gastric epithelium. Some factors contribute for this fact: enzymes as Mucinases, proteases, lipases and urease, Lipopolysacharides (LPS), toxins as Vacuolization cytoxin A (VacA) and cytotoxin associated gene A (Cag A) [1,2]. Mucinases, proteases and lipases are bacterium enzymes that permit, respectively, degradation of the gastric mucus, protein and lipids, facilitating bacterium proliferation and epithelium colonization. The main enzyme responsible for H. pylori survival in gastric ambient is urease. Urease is an enzyme capable of convert urea of the cells in ammonia and CO2. Ammonia is a component with basic pH that permit that acid gastric pH be neutralized (Figure 1A & 1E).

This changed gastric environment facilitates, together other factors of virulence, tissue infection by the bacterium. The immunologic cells quickly activate your defense mechanism. The Lipopolysacharides (LPS) is a structural factor that difficult immunologic activity and permit bacterium immunologic evasion [3]. Even so the inflammatory acute answer is able to cause neutrophil infiltration and glandular aggression in gastric mucosa (Figure 1B & 1E). This is the first step in a sequence of factors that will be associated with the development of mucosa

atrophy and atrophic gastritis (Figure 1C & 1E). VacA and CagA are invasive toxins associated to physiopathology of the gastritis. CagA cause cytoskeletal changes, interfering in microtubules associated protein (MAP). This mechanism alters cytoskeletal organization, causing cell disorganized proliferation and disruption of the junction structures, as E-cadherin. E-cadherin and β-catenin form one structure complex in cell membrane. Changes in e-cadherin and the damages by CagA are able to cause intracellular accumulation of β-catenin. Consecutive effects of this accumulation are the methylations of some genes as CDX1, CDX2 AND MUC2, associated to intestinal metaplasia development (Figure 1D & 1E).

The Continue exposition to toxin can activate other genes methylation, as MLH1, APC, COX2, erbB2 AND TP53, favoring the dysplasia and neoplasm formation (Figure 1E) [1]. VacA is other toxin that permit mythocondrial vacuolization and apoptosis. Cell death culminate in exposition of the cell antigens as ATPase H+/K+ and cytochrome C, main antigens responsible for encourage B lymphocyte differentiation in plasma cell, with antibody productions, one chronic process that also have how final consequence atrophy of the mucosa [1]. Lymphocyte infiltration, in some cases, culminates in lymphoid aggregates. These aggregates of neoplasm are also known as lymphoma. This is the resume of physiopathology of the H. pylori bacterium in the gastric tissue.

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