Our results show that HtrA is highly conserved among clinical isolates, reinforcing its essentiality for H. pylori survival.


Abstract no.: P03.09

REGULATION OF MDM2 ONCOGENE BY HELICOBACTER PYLORI LIPOPOLYSACCHARIDE IN GASTRIC EPITHELIAL CELLS

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Purpose: Mdm2 is critical regulators of the p53 protein which plays a crucial role in maintaining genomic integrity and tumor prevention. Helicobacter pylori is reportedly involved in the development of gastric cancer. We investigated the mechanisms between H. pylori and MDM2, focusing on H. pylori-derived lipopolysaccharide (LPS).

Experimental Design: H. pylori-LPS and two gastric cancer cell lines (AGS and MKN28) were used. We examined whether the expression of MDM2 in a dose- and time-dependent manner of gastric epithelial cells, when they are exposed to H. pylori-LPS. We also examined if PI3K/Akt/mTOR signaling pathway mediated this expression. Western blotting was employed to evaluate the expressions of MDM2, p-Akt-S473 and Akt, and the functionality of the MDM2 promoter is examined by luciferase assay.

Results: Gastric epithelial cells express more MDM2 in a dose- and time-dependent manner when they are stimulated with H. pylori-LPS. Treatment of gastric epithelial cells application of LY294002 and Rapamycin caused a dramatic reduction of H. pylori-LPS induced MDM2. In addition, H. pylori-LPS stimulation increased the MDM2 promoter activity.

Conclusion: H. pylori-LPS induced MDM2 over expression is mediated by PI3K/Akt/mTOR.

Abstract no.: P03.10

DISRUPTION OF TIGHT JUNCTIONS OF GASTRIC EPITHELIAL CELLS INDUCED BY HELICOBACTER PYLORI AS ANALYSED USING REAL-TIME PHASE CONTRAST MICROSCOPY

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Helicobacter pylori cytotoxin-associated gene A (CagA) has been regarded as a major player in the disruption of tight junctions. However, the exact mechanism of tight junction disruption induced by H. pylori is still not well-established. This study uses a high resolution imaging system that is able to maintain perfect focus and optimal growth conditions for cells to follow live cell observations. Using MKN28 cells, which form functional tight junctions, these cells were infected with H. pylori phylogenetically diverse LPS-induced MDM2 over expression is mediated by PI3K/Akt/mTOR. The real-time event of tight junction disruption as shown by the real-time microscopic observations is further supported by results obtained from barrier function test. Taken together, our findings show that real-time phase contrast microscopy can provide a highly supportive role on the mechanistic events occurring during host-pathogen interactions.

Abstract no.: P03.11

ULCEROGENIC PROFILE OF HELICOBACTER PYLORI PEDiatric STRAINS: A CONTRIBUTION TO GET INSIGHT INTO THE VIRULENCE OF THE BACTERIA

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Helicobacter pylori infection is the major cause for the development of peptic ulcer disease (PUD). In addition to patient genetic susceptibility, PUD occurrence in...