Abstract no.: P3.5
DIFFERENTIAL EXPRESSION OF HUMAN BETA DEFENSIN- 2 AND -3 IN GASTRIC MUCOSA OF HELICOBACTER PYLORI-INFECTED INDIVIDUALS
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Background: Antimicrobial peptides are keyplayers of initial innate immune responses to human pathogens. Two major representatives, the human beta defensins 2 and 3 (hBD2, hBD3) are both known to be induced by Helicobacter pylori. Previously, it was demonstrated in vitro that H. pylori actively abrogates hBD3 expression during prolonged infections. Here we comprehensively assessed hBD2 and hBD3 expression ex vivo in the gastric mucosa of healthy individuals.

Materials and Methods: Twenty volunteers (H. pylori positive and H. pylori negative: n = 10) were enrolled. H. pylori positive subjects underwent eradication therapy and repeated the protocol. Expression of both defensins were assessed by quantitative RT-PCR and ELISA, and correlated with histopathological degree of gastritis.

Results: hBD2 and hBD3 were found to be ubiquitously expressed in all three groups. In general, hBD2 mRNA levels were significantly decreased, while corresponding protein levels remained unchanged. Eradication therapy led to normalization of mucosal hBD2 expression, while hBD3 expression demonstrated high interindividual variations among individuals.

Conclusions: Both defensins are ubiquitously but differentially expressed in gastric mucosa in relation to H. pylori infection. Ex vivo data support previous in vitro findings that H. pylori infection is associated with reduced hBD3 expression in chronic active gastritis.

Abstract no.: P3.6
BLOOD AND LYMPHATIC MICROVESSELS DENSITY IN GASTRIC MUCOSA OF DYSPHASIC PATIENTS
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Introduction: Published data on the role of H. pylori in angiogenesis are contradictory, while its role in lymphangiogenesis has not been investigated.

Aims: To investigate the density of lymphatic and blood vessels, together with H. pylori infection, in biopsies of gastric body and antrum of dysphasic patients.

Methods: Biopsies from patients subjected to gastroscopy according to clinical criteria were studied by immunohistochemistry. Exclusion criteria were: previous eradication therapy, ulcer disease, regular non-steroidal anti-inflammatory use, and proton pump inhibitor or antibiotic treatment 15 and 30 days prior to gastroscopy. MicrovesSEL density was determined with CD31 and D2.40 monoclonal antibodies (DAKO, Glostrup, Denmark), which are markers of blood and lymphatic vasculature respectively. Quantification was performed by direct counting of microvessels in four fields at 40· magnification. H. pylori infection was assessed by ¹³C-urea breath test and/or histopathological diagnosis.

Results: Twenty-three biopsies (13 antral) from 13 patients were studied. Their median age was 62 years, 46% males, 73% gastritis and/or atrophy and 24% H. pylori positive. Higher blood microvesSEL density was associated to H. pylori infection (p = 0.03) (Table 1). MicrovesSEL count was slightly elevated, without statistical significance, among biopsies with histological diagnosis of gastritis. Further associations with inflammatory activity or location were not found. Lymphangiogenesis was not related with any of the studied variables.

Conclusions: (1) H. pylori infection was associated with increased gastric angiogenesis (2) Lymphangiogenesis was not related with the studied clinico-pathological features.

Abstract no.: P3.7
COX-2 INHIBITION WITH NUTRATEUTICALS: A NEW THERAPEUTIC APPROACH AGAINST HELICOBACTER PYLORI INFECTION?
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Background: Immunizing chickens with certain antigens and collecting the antibodies with polyclonal antibodies in the chicken eggs. Colorimetric reaction of anti-Helicobacter pylori antibodies with Helicobacter pylori···specific antigens was not observed.

Materials and Methods: We infected 45 C57BL/6 mice with SS1 – H. pylori strain. After infection confirmation by ¹³C-urea breath test mice where then treated with either PBS, curcumin (10 mg/mouse) or Symbiotic 2000° (50 mg/mouse), three times per week. Five mice from each treatment group were euthanized at week 6, 18 and 27. Gastric samples were removed for COX-2 immunohistochemistry analysis.

Results: All the 45 mice were Hp positive by ¹³C-urea breath test and immunohistochemistry. In the PBS group the production of COX-2 was significantly up-regulated at week 6 (area of positive immunostaining 242–614 × 10³ pixels) and 18 (area of positive immunostaining 242–614 × 10³ pixels) and 27 week (area of positive immunostaining 129–175 × 10³ pixels). The treatment with either curcumin or symbiotic significantly decreased the expression of COX-2 at all time points.

Conclusions: These results suggest the therapeutic usefulness of both nutrateuticals on COX-2 inhibition during chronic experimental mice H. pylori infection. The supplementation of diet in humans with curcumin or Symbiotic 2000™ may be a novel therapeutic approach against gastric inflammation induced by Hp infection.