which may act as potential markers for neoplastic diseases a may be used in specific serological tests. Immunoproteome assay was used to identify *H. pylori* antigens, recognized by sera from patients with peptic ulcer, bleeding peptic ulcers, gastric cancer, and dyspepsia. We performed proteomic maps of *H. pylori* strain 23Ca3 (patient with gastric cancer), probed against single sera from three groups of *H. pylori* -positive patients (peptic ulcer, gastric cancer, and dyspepsia). Immunoreactive spots were identified by LC/NSI-MS/MS. In this study, we detected eleven immunoreactive spots with the sera from three groups of patients. 50S ribosomal protein L7/L12 was the only proteína recognized by the three groups of sera, which highlights it as a protein useful in the diagnosis of *H. pylori* infection regardless of the pathology in the stomach. Additionally, we found proteins that share recognition in sera from patients with gastric cancer and dyspepsia. These immunoreactive spots may be promising for developing specific serological tests to differentiate patients with gastritis at high risk for gastric cancer, to be evaluated in prospective investigations.

Abstract no.: P11.06

THE INFLUENCE OF BLOCKING TIM-3 SIGNAL PATHWAY ON IMMUNE PROTECTION OF *H. PYLORI* VACCINE AND TH IMMUNE RESPOND

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Objective: To observe the influence of blocking Tim-3 signal on immune protection of Hp vaccine and Th respond.
Methods: BALB/c mice were divided into three groups and immunized by: 1. Control group; 2. Hp vaccine; 3. Anti-Tim-3 antibody pretreatment + Hp vaccine; At 4 weeks after the last immunization, the mice from 1 and 3 groups were challenged by Hp quartic. At 4 weeks after the last challenge, mice were sacrificed and sample were collected. Hp, the level of cytokine, Foxp3$^+$Treg in gastric mucosa were determined.
Results: 1, Hp colonized was significantly lower in group with anti-Tim-3 antibody pretreatment than that in group without pretreatment ($p < .05$). 2, Iinflammatory of mice of Hp vaccine was higher than that in control ($p < .01$), and in group with anti-Tim-3 antibody pretreatment were higher than group without pretreatment ($p < .05$). 3, The level of of Th1 cytokine in mice of Hp vaccine were significantly higher than that in control ($p < .05$), and in group with anti-Tim-3 antibody pretreatment were significantly higher than those in groups without pretreatment ($p < .05$); The level of Th2 cytokine in mice, there were no significantl difference among all groups ($p > .05$). 4, The Foxp3$^+$Treg in mice of Hp vaccine were significantly higher than that in control ($p < .01$), and in group with anti-Tim-3 antibody pretreatment were significantly lower than those in groups without pretreatment ($p < .01$).
Conclusion: Blocking Tim-3 signal pathway can improve the Hp vaccine protection and promote Th1 immune respond and reduced the numbers of CD4+CD25+Foxp3+Treg, this could be the mechanism that it enhanced Hp vaccine immune protection.

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THE INFLUENCE OF BLOCKING CD25 ON IMMUNE PROTECTION OF *H. PYLORI* VACCINE AND TH IMMUNE RESPOND

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Objective: To observe the influence of blocking CD25 on immune protection of Hp vaccine and Th respond.
Methods: BALB/c mice were randomly divided into three groups: 1. Control; 2. Hp vaccine; 3. Anti-CD25 antibody pretreatment + Hp vaccine. At 4 weeks after immunization, mice from 2 and 3 groups were challenged by Hp. At 4 weeks after challenge, Hp, the level of cytokine, Foxp3+Treg in gastric mucosa were determined.
Results: 1, Hp colonized was significantly lower in group with anti-CD25 antibody pretreatment than that in group without pretreatment ($p < .05$). 2, Inflammatory degree in mice of Hp vaccine was higher than that in control ($p < .01$), and in group with anti-CD25 antibody pretreatment were higher than group without pretreatment ($p < .05$). 3, Level of Th1 and Th17 cytokine in mice of Hp vaccine were significantly higher than that in control ($p < .05$), and in group with anti-CD25 antibody pretreatment were significantly higher than those in groups without pretreatment ($p < .05$); Level of Th2 cytokine, there were no significantl difference between in control and vaccine ($p > .05$), and between in group with anti-CD25 antibody pretreatment and without pretreatment ($p > .05$). 4, Foxp3+Treg in mice of Hp vaccine were significantly higher than that in control ($p < .01$), and in group with anti-CD25 antibody pretreatment were significantly lower than those in groups without pretreatment ($p < .05$).
Conclusion: Blocking CD25 can improve the Hp vaccine protection and exacerbate the inflammation in mice of Hp vaccine; and can promote Th1 and Th17 respond and reduced Foxp3+Treg, this could be mechanism that it enhanced Hp vaccine immune protection.

Abstract no.: P11.08

NUTRACEUTICALS: A NEW THERAPEUTIC APPROACH AGAINST *HELICOBACTER PYLORI* INFECTION?

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Background and Aim: *H. pylori* induces severe gastric chronic inflammation and is the cause of gastritis, peptic ulcer and a major risk factor for gastric cancer. The aim of the study was to investigate the anti-inflammatory effect of two nutraceuticals in Hp-infected mucosa.
Materials and Methods: Eighteen C57BL/6 mice were inoculated with Hp SS1 by gavage three times with 3×10^9 viable cells. Mice were then treated with either PBS, curcumin (10 mg/mouse) or Symbiotic 2000® (50 mg/mouse), three times per week. Half of the infected and three non-infected mice were euthanized at week 6, the remaining at week 18. Gastric samples were removed for immunohistochemistry and PCR array (inflammatory response and immunity pathway) analysis (Sabiosciences, Qiagen).
Results: All the 18 mice were Hp positive by immunohistochemistry. The production of the chemokines CCL2, CCL5, CCL20, CCL25, CXCL1 and CXCL11 was significantly up-regulated at both week 6 (range of fold-change 4.3–718) and week 18 (range of fold-change 16–1192). Similarly, the expression of the proinflammatory cytokines IL-1β, IL6, IL9, IL10, IL23, TNFα and INFγ was significantly augmented (range of fold-change 1338–8251). The treatment with either curcumin or symbiotic drastically decreased the expression of all these mediators, restoring their levels to those similar to the non-infected mice.
Conclusions: The present study confirmed that Hp infection induces a strong inflammatory response. Curcumin and Symbiotic treatments exerted a significant anti-inflammatory effect in Hp-infected mucosa.
The supplementation of diet with these nutraceuticals may be a novel clinical approach against gastric inflammation induced by Hp infection.

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HELICOBACTER PYLORI INFECTION: THE ROLE OF INTESTINAL MICROBIOTA MODULATION

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Intestinal microbiota may influence inflammation in the host. The aim of the present study was to explore the role of modulation of intestinal microbiota in the outcome in the *Helicobacter pylori* (Hp) gastric inflammation.
Twenty five C57BL/6 male mice were separated in three groups: Control group (CG) n = 5 Infected group (IG) n = 10 and Synbiotic 2000™ (SG) n = 10. CG received PBS by gavage; IG and SG were inoculated intragastrically with *H. pylori* SS1 cell suspension (10^9 CFU/mL). Then, mice were treated either with PBS (CG and IG) or Synbiotic 2000™ (SG). Five mice from each group were sacrificed at week 6 and the other at week 18. At each time samples were collected from: gastric tissue to immunohistochemistry and histological evaluation (HE) and faeces to evaluate intestinal microbiota composition by FISH, targeting 14 bacterial groups.
IG and SG groups were *H. pylori* positive by immunohistochemistry. Microbiota analysis: In IG there were significant changes in the microbiota composition, comparing to CG. At week 6 there were changes in 12 of 14 (85.7%) bacterial groups, while at week 18 there was a change in 6/14 (42.9%). In SG, there were changes in 7/14 (50.0%) at week 6, and in 4/14 (28.6%) at week 18, comparing to CG. Histology: IG at weeks 6 and 18 has 40% (2/5) of intramucosal inflammation and SG at the same end points has 0% (0/5).
These results suggest that modulation of the intestinal microbiota by Synbiotic 2000™ may influence the outcome of Hp gastric inflammation.

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