Maternal antibiotic treatment peripartum has differential consequences on protein expression of intestinal cytoprotective heat shock proteins in her offspring

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Antagonistic Effects of Probiotic E. coli Nissle 1917 on Various EHEC Strains

Satoshi Inukai, Gail A. Hecht

Introduction: To the continental application and extensive studies of its efficacy and molecular biology, E. coli Nissle 1917 (EcN) is among the best characterized probiotics. EcN is approved as a pharmaceutical in the EU and widely used in treatment of gastrointestinal disorders. In 2011, pathogenic Shiga toxin-producing E. coli O104:H4 caused the largest outbreak of enterohemorrhagic E. coli (EHEC) recorded so far. In this study, the influence of the probiotic E. coli Nissle strain 1917 on adhesion and growth of pathogenic E. coli strains (pEc) was investigated in vitro. We used EHEC (O157:H7), enterogaugative E. coli (EAEc, O104:H4), enterohemorrhagic E. coli (EPEC, O153:H31), and two O104:H4 EHEC (clinical isolates) from the recent outbreak in Germany in our experiments. Shiga toxin production of the EHEC strains was elucidated in co-culture with EcN. We are currently investigating the mechanisms responsible for these important antagonistic effects of EcN on pEc strains. Methods: 24-well plates were coated with the human gut epithelial cell lines Caco2 or IPEC-J2, to determine the adhesion of living bacteria to these cells. Single cultures of E. coli were incubated for 30 minutes with Caco2 or IPEC-J2 and then the number of adherent bacteria were counted with a microscope. The plates were then washed with the epithelial cells, following by washing, epithelial cell lysis and plating of serial dilutions of the resulting bacterial suspension on agar plates. Furthermore, the growth of the bacteria in each well was determined at t=0h, t=2h, t=18h and t=24h by plating of serial dilutions on agar plates. Shiga toxin production of single cultures of E. coli EHEC, and co-cultures at ratios of 1:1 and 10:1 (EcN:EHEC) was analyzed after 24h incubation via Verotoxin ELISA.

Results: EcN significantly reduced the adhesion of all tested pEc strains to Caco2 and IPEC-J2 by up to 59% for E. coli O104:H4, UPEC; O153:H31, EHEC O157:H7 and to >80% for O104:H4 EHEC, in a co-culture with 10-fold EcN. In addition, inhibition of growth, due to killing of pathogenic E. coli, occurred. The number of EHEC (O157:H7) in co-culture with 10-fold EcN decreased at a ratio of 1:1 (EcN:EHEC) by ≥90%. Microcins were identified as the first factor contributing to the antagonistic effects of EcN on pEc strains. Discussion: In our experiments reduction of Shiga toxin production and adhesion to human gut epithelial cell lines, as well as inhibition of EHEC growth was observed. This is a good indication that EcN might be useful in preventing in infections with EHEC strains and even assist in the treatment of acutely affected patients. However, more experiments and clinical trials have to be conducted before EcN can be considered as a treatment option for EHEC infections.

Gut Microbiota Urease Activity Regulates Serum Urea Levels on a Low Protein Diet

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Background: The gut microbiota is one of the most densely populated bacterial communities on earth and plays an important role in the normal metabolic homeostasis of the mammalian host. Classic studies in physiology have shown that it helps to maintain host nitrogen balance through the hydrolysis of colonic urea and the subsequent absorption of resultant ammonia. Reduction in the consumption of protein due to illness, as part of medical therapy, or as a result of lifestyle factors, can affect the abundance of some gut bacteria. In a prior study, we examined the role of the gut microbiota on metabolic disturbances associated with the consumption of a low protein diet (LFD). Methods and Results: Female C57Bl/6j mice were fed either a normal chow (20% kcal protein) or LFD (3% kcal protein) for 1 month. Metabolic phenotyping revealed that LFD mice failed to gain weight and suffered from a profound defect of water conservation manifested as severe polydipsia and polyuria. This diet was due to the inheritance of hepatic ureagenesis and the reduction in serum urea levels. 16S rRNA gene sequencing revealed that the gut microbiota was altered significantly by the LFD but in a manner that was not directly associated with this metabolic phenotype. Reduced antibiotic concentrations of the gut microbiota to LFD restored serum urea levels and prevented the development of polydipsia and polyuria. Antibiotic-induced restoration of serum urea levels did not alter host ureagenesis in LFD mice since hepatic urea cycle gene expression remained suppressed. By contrast, a 1C-urea breath test and direct measurement of oral urea activity revealed that oral antibiotic treatment dramatically reduced colonic urea hydrolysis thereby helping to preserve circulating host urea levels. Interestingly, the 1C-urea breath test also showed that colonic urease activity was reduced by a LFD demonstrating the impact of diet on gut microbiota function. Conclusions: Our results demonstrate that oral antibiotics alter the host-microbial urea equilibrium in mice fed a LFD by reducing the capacity of the gut microbiota to hydrolyze host-produced urea. When ureagenesis is suppressed by a LFD, serum levels of urea fall as the gut microbiota continues to consume urea in the colon. Antibiotic treatment of LFD mice restores urea serum levels by reducing the hydrolysis of colonic urea thereby disrupting the diffusion gradient and reducing the movement of urea into the colon. Finally, alterations in gut microbiota composition as well as the reduction of its urease activity on a LFD indicate that diet alters not only the structure but also the function of the gut microbiota. This model system may prove to be a powerful tool in assessing interventions that modulate gut urease activity for the treatment of disease.

Maternal Antibiotic Treatment Peripartum Has Differential Consequences on Protein Intake of Intestinal Cytotoxic Tissue Protein Expression in Her Offspring

Emre Ersan Erdogan, Haluk Erdogan

Background: Peripartum antibiotics are thought to have negative consequences on intestinal barrier function, immune system development and long-term health. These effects might be related to disturbances in neonatal bacterial colonization. Protective heat shock proteins (HSPs) are induced by induced physiological and pathological stress in intestinal epithelial cell (IEC) and in mouse intestine. HSP90 has been recently shown to contribute to IEC protection in vitro but data in vivo are lacking. We aimed to test the hypothesis that early alterations in gut microbial colonization impact intestinal cytototoxic HSPs. Methods: Sows received amoxicillin orally (10mg/kg BW, ATBQ n=11; vs. untreated CTL, n=12)
Neonatal Probiotic Administration Has Long-Lasting Effects on Gut Permeability Responses to Stress in Adult Pigs Born to Sows Treated With Antibiotics Around Parturition
Jean-Paul Lalles, Dominique Bertaccini, Gerard Savary, Hauke Smidt
Background: Peripartum antibiotics are thought to have long-lasting negative consequences on intestinal barrier function, immune system development and long-term health, e.g. allergy. Various kinds of stress (e.g. nutrition, environment) can be deleterious to gut barrier function. We have designed the hypothesis that antibiotic administration alter various facets of gut barrier in their offspring during development and in adulthood. We tested with this model the hypothesis that early administration of a probiotic to offspring can modulate gut barrier function in young adults depending on diet and stressor. Methods: Sows (n=20) received amoxicillin orally (40 mg/kg BW/d) around parturition (d-10 till d+21). Offspring (1/litter) were sacrificed during the suckling period (d14, d28 and d42) and ileal tissue were collected from the proximal colon around parturition by laparotomy. Intestinal HSP expression. Changes in offspring intestinal microbiota and long-term effects of perinatal antibiotic treatment on offspring intestinal HSF family are being investigated.

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Chronic Ingestion of Lactose: Malabsorber Host and Intestinal Microbiota Adaptations
Virginie Alexandre, Amaury Lang, François Bacher, Robert Benamouzig, Yolanda Sanz, Daniel Tomé, Anne-Marie Davila
Introduction: Physiological and metabolic consequences of chronic lactose ingestion by malabsorbers are poorly described. In order to highlight the suspected maladaptation, adaptations, intestinal microbiota and host’s physiology have been in vivo studied in hypolactasic rats, over a prolonged ingestion of lactose. Materials and methods: Two groups of Wistar male rats ingested a diet containing 25% of lactose (L25 group) or 25% of sucrose (para-lactose group) (PL group). Consecutive slaughters were performed over 6 days of the diet. Bacterial groups’ evolutions (qPCR), short-chain-fatty-acids (SCFA) concentrations and βgalactosidase activity were evaluated in caecal and colonic contents. Concomitantly, SCFA were quantified in portal blood, myeloperoxydase activity (MPO) was evaluated in colonic mucosa and histological samples were analysed. Results: L25 vs PL comparisons over time reveal a decrease in portal blood, myeloperoxydase activity were evaluated in caecal and colonic contents. Concomitantly, SCFA were quantified in portal blood, myeloperoxydase activity (MPO) was evaluated in colonic mucosa and histological samples were analysed. Results: L25 vs PL comparisons over time reveal a decrease in portal blood, myeloperoxydase activity were evaluated in caecal and colonic contents. Concomitantly, SCFA were quantified in portal blood, myeloperoxydase activity were evaluated in caecal and colonic contents. Concomitantly, SCFA were quantified in portal blood, myeloperoxydase activity were evaluated in caecal and colonic contents. Concomitantly, SCFA were quantified in portal blood, myeloperoxydase activity were evaluated in caecal and colonic contents. Concomitantly, SCFA were quantified in portal blood, myeloperoxydase activity were evaluated in caecal and colonic contents. Concomitantly, SCFA were quantified in portal blood, myeloperoxydase activity were evaluated in caecal and colonic contents. Concomitantly, SCFA were quantified in portal blood, myeloperoxydase activity were evaluated in caecal and colonic contents. Concomitantly, SCFA were quantified in portal blood, myeloperoxydase activity were evaluated in caecal and colonic contents. Concomitantly, SCFA were quantified in portal blood, myeloperoxydase activity were evaluated in caecal and colonic contents. Concomitantly, SCFA were quantified in portal blood, myeloperoxydase activity were evaluated in caecal and colonic contents. Concomitantly, SCFA were quantified in portal blood, myeloperoxydase activity were evaluated in caecal and colonic contents. Concomitantly, SCFA were quantified in portal blood, myeloperoxydase activity were evaluated in caecal and colonic contents. Concomitantly, SCFA were quantified in portal blood, myeloperoxydase activity were evaluated in caecal and colonic contents. Concomitantly, SCFA were quantified in portal blood, myeloperoxydase activity were evaluated in caecal and colonic contents. Concomitantly, SCFA were quantified in portal blood, myeloperoxydase activity were evaluated in caecal and colonic contents. Concomitantly, SCFA were quantified in portal blood, myeloperoxydase activity were evaluated in caecal and colonic contents. Concomitantly, SCFA were quantified in portal blood, myeloperoxydase activity were evaluated in caecal and colonic contents. Concomitantly, SCFA were quantified in portal blood, myeloperoxydase activity were evaluated in caecal and colonic contents. Concomitantly, SCFA were quantified in portal blood, myeloperoxydase activity were evaluated in caecal and colonic contents. Concomitantly, SCFA were quantified in portal blood, myeloperoxydase activity were evaluated in caecal and colonic contents. Concomitant