Methods: Equine jejunal epithelia were mounted in Ussing chambers and ischemia was simulated (1% O_2). Part of the epithelia was incubated with 1 mM $CoCl_2$. Viability was evaluated by measuring the increase of short circuit current after mucosal addition of 2 mM glucose. Fluxes of ^{3}H-mannitol were measured. mRNA expression of cyclooxygenase 1 and 2, tumour necrosis factor α, hypoxia inducible factor 1α and intestinal trefoil factor was assessed by RT-qPCR.
Results: Short circuit current was decreased by ischemia compared to normoxia and showed no tendency to recover. In contrast to that, fluxes of ^{3}H-mannitol showed no alterations. Cyclooxygenase 2 mRNA expression was increased after ischemia plus reperfusion. Incubating epithelia with 1 mM $CoCl_2$ tended to reduce this increase while short circuit current was decreased by addition of $CoCl_2$ even under normoxia.
Conclusions: Our data indicate a severe damage of the enterocytes after ischemia. There are signs for an activation of cyclooxygenase 2 pointing to an involvement of nuclear factor κB. The role of hypoxia inducible factor 1 and nuclear factor κB will be further investigated in Western Blot studies.
Keywords: ischemia, jejunum epithelium, mRNA-expression.

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Expression of microbiota recognition receptors and intestinal serotoninergic system in two models of colitis

Elena Layunta[1], Eva Latorre[1], Laura Grasa[1], Marta Castro[1], Miguel-Angel Plaza[1], Juan Pardo[2], Lourdes T Santiago[2], Fermin Medina de Sánchez[1], Olga Martínez-Augustin[4], Borja Ocón[3], Clara Aranda[4], José E Mesonero[1], Ana I Alcalde[1]
[1]Department of Pharmacology and Physiology, University of Zaragoza, Spain; [2]Department of Biochemistry and Molecular and Cellular Biology, University of Zaragoza, Spain; [3]Department of Pharmacology, CIBERehd, University of Granada, Spain; [4]Department of Biochemistry and Molecular Biology II, CIBERehd, University of Granada, Spain

Background: The intestinal microbiota patterns recognition receptors TLRs and NODs (PRRs), and the intestinal serotoninergic system, may contribute to intestinal responses to microbiota and alter intestinal homeostasis/inflammation e.g. in inflammatory bowel disease.
Aim: We examined two mouse colitis models (Dextran Sulfate Sodium (DSS) or Lymphocyte Transfer (LT) mouse colitis) and compared the expression of PRRs, implicated in innate immunity, and some elements of the intestinal serotoninergic system.
Methods: In ileum and colon from DSS or LT mouse colitis animals, TLRs, NODs, serotonin transporter (SERT) and serotonin receptors (5-HTRs) mRNA expression was measured by RT-qPCR. SERT protein expression was analyzed by western blotting.
Results: In DSS ileum, TLR9, 5HTR1A, 5-HTR4 and 5-HTR7 mRNA levels were over-expressed, and SERT expression reduced; in DSS colon, NOD2, TLR2, TLR9, and 5-HTR7 mRNAs were increased; however, 5HTR1A, 5-HTR2B, 5-HTR3, and 5HTR4 mRNA levels were diminished, as well as SERT expression. On the other hand, in LT ileum, TLR9, 5-HTR1A, 5-HT2A, and 5-HT2B mRNAs were reduced and, although SERT mRNA was not altered, SERT protein level was reduced; in colon of LT mouse model, TLR2, TLR9, 5-HTR1A, and 5-HTR7 mRNA levels and SERT expression were increased; however, TLR4, NOD1, 5-HTR2B, 5-HT3, 5-HT4 mRNA levels were reduced.
Conclusions: These results highlight the complexity of the participation of intestinal serotoninergic and innate immune system in both colitis models.

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Keywords: Colitis, intestine, NOD, serotoninergic system, TLR.

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Intestinal sugar transporters as sensors for incretin secretion: studies in mice and murine gut organoids

Tamara Zietek[1], Eva Rath[2], Pia Röder[1], Dirk Haller[2], Hannelore Daniel[1]
[1]Biochemistry, ZIEL Research Center of Nutrition and Food Sciences, Technische Universität München, Freising, Germany; [2]Biofunctionality, Technische Universität München, Freising, Germany

Background: It was recently shown that the sodium-dependent glucose transporter SGLT1 is involved in intestinal glucose sensing and mediates the secretion of the incretin hormones glucagon-dependent insulinotropic polypeptide (GIP) and glucagon-like peptide-1 (GLP-1). Furthermore, the glucose transporter GLUT2 was proposed to contribute to glucose sensing. Fructose also induces GLP-1 secretion, however the responsible sensor has not been identified yet.
Aim: To assess the role of the intestinal sugar transporters SGLT1, GLUT2 and GLUT5 in sugar transport and sensing in mice and murine intestinal organoids.
Methods: Incretin and insulin responses to an oral glucose- or fructose-load were measured in plasma samples of wild type (WT) mice and mice lacking either SGLT1, GLUT2 or GLUT5. GIP and GLP-1 secretion in response to glucose or fructose was measured in pri-

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