

the activation of the innate immune response. Currently, few data are available on the interactions between human skin cells and USUV as well as on the inflammatory and antiviral responses induced. In order to study the pathophysiology of USUV skin infection, the permissivity of resident skin cells to USUV infection and the pathways involved in viral recognition and innate immune response activation were characterized. Our results show an early viral replication during the first 24 hours in human primary keratinocytes and fibroblast associated with the production and the induction of pro-inflammatory targets. In *ex vivo* human skin explants, viral replication was observed 24 hours post-infection in epidermis and dermis. In addition, using a mouse model subcutaneously infected by USUV in the back, the results showed the permissiveness of murine skin cells to USUV infection as well as the dissemination of the virus from the inoculation site to the distal skin tissues associated with a persistence of the infection up to 6 days post-infection in all tissues. Finally, a prominent role of the RIG-I receptor in the recognition of USUV and in the induction of the inflammatory and antiviral response leading to the restriction of the infectious viral particle production was demonstrated. Together, these data provide a better understanding of the pathophysiology of the early stages of USUV skin infection.

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Hydrogen sulfide-producing *Escherichia coli* strains in a patient with Crohn's colitis: a clinical case

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Crohn's disease (CD) is thought to result from a combination of factors including altered genetics and environmental factors affecting the gut microbiota. To promote a better understanding of host–microbiota interaction, we cultured fecal bacteria from 14 CD patients. Here we report a clinical case of a CD patient (male, 53 years old) with colitis colonized by three *Escherichia coli* strains producing hydrogen sulfide (H₂S). The gas production is not a typical characteristic of the *E. coli* species. High concentration of H₂S in gut leads to colonic mucosa damage and increases its permeability. Whole-genome sequencing was performed to characterize the isolated fecal H₂S-producing strains using Illumina NextSeq 500 platform. *E. coli* draft genome assemblies were submitted to NCBI (GCA_008040715.1, GCA_008040625.1, GCA_008040635.1). Genome analysis revealed that the strains carried 3-mercaptopyruvate sulfurtransferase gene, required for the H₂S production. Colonic bacteria are known to produce H₂S when exposed to antibiotics and oxidative stress. However, two isolated *E. coli* strains turned out to be resistant only to cephalosporins (cefazolin and/or cefotaxime) and the third strain was sensitive to all tested antibiotics (tetracycline, amikacin, meropenem, nalidixic acid, ciprofloxacin, levofloxacin, moxifloxacin, aztreonam, ampicillin, sulfanilamide, cefazolin, cefepime, cefotaxime, cefuroxime). So the isolated H₂S-producing *E. coli* strains were sensitive to a range of antibiotics, tolerate oxidative stress in the gut, and their defense mechanisms may contribute to intestinal inflammation. This work was supported by the Kazan Federal University Strategic Academic Leadership Program (PRIORITY-2030) and by the subsidy

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Rs2274910 in ITLN1 gene is associated with reduced levels of short-chain fatty acids in the feces of patients with Crohn's disease

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The variant rs2274910 in the ITLN1 gene is associated with higher risk of Crohn's disease (CD) development. This gene encodes intelectin-1, which is produced by goblet cells and secreted into the intestinal lumen. Intelectin-1 recognizes and binds specific carbohydrate-containing structures of microbial glycans and lipopolysaccharides, thereby activating bacterial phagocytosis. Thus, ITLN1 modulates the interaction between the gut microbiota and the host. The gut microbiota produces a number of metabolites, including short-chain fatty acids (SCFAs), which are an energy source for intestinal epithelial cells and regulate proliferation, differentiation, and immune response. In our study, we performed the genotyping analysis of the rs2274910 by MALDI-TOF minisequencing and determination of fecal SCFAs (acetic, propionic, butyric, isobutyric, valeric, isovaleric, caproic, isocaproic) content by gas chromatography for 66 patients with CD. Spearman's rank correlation coefficient between SCFAs concentrations and CD patients genotypes was calculated using an additive model (depending on the genotype, a higher risk of developing CD corresponds to a higher rank). Statistically significant negative correlations were found between rs2274910 and the levels of propionic ($R = -0.31$), butyric ($R = -0.26$), valeric ($R = -0.30$) and caproic ($R = -0.26$) acids ($P < 0.05$). These observations may suggest that in CD patients with a risk variant in the ITLN1 gene, intelectin-1 recognizes carbohydrates on the surface of beneficial microorganisms that produce SCFAs, activating their uptake by macrophages. This can cause dysbiotic changes in the intestinal microbiota composition and the progression of inflammatory bowel diseases. The work was supported by the Kazan Federal University Strategic Academic Leadership Program (PRIORITY-2030) and by the subsidy allocated to Kazan Federal University for the state assignment in the sphere of scientific activities (#FZSM-2023-0013).

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Unravelling the novel role of staphylococcal superantigen-like protein in amyloid-based biofilm formation

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Staphylococcus aureus is a highly dangerous pathogen that represents a significant burden on the current healthcare system.