Conclusion. The intestinal barrier function in piglets plays a critically important role in maintaining organism homeostasis. To evaluate the effectiveness of preparations, it is promising to use a combined strategy for determining specific molecular markers of barrier function, the state of the intestinal microbiome, morphological indicators of the intestinal epithelium, immune response markers, and productive indicators.

ISOTONIC PROTEIN FORMULATION INHIBITS PEDV PROLIFERATION IN PIGLETS VIA MODULATION OF ENTEROCYTE JUNCTIONS AND INTERFERON PRODUCTION

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Abstract. The porcine epidemic diarrhea virus (PEDV) not only causes large economic losses in the swine industry, as the causative agent of PED, but also poses a risk to other animals and human due to cross-species transmission. Piglets are particularly susceptible to PEDV infection, which disrupts intestinal epithelium morphology and barrier integrity as well as nutrient absorption. PEDV is one of the most important swine viruses that has emerged or re-emerged, posing a significant threat to the global pork industry. In particular, the highly pathogenic PEDV strain, which began to spread in China in 2013, emerged in the United States and then spread to Asian countries such as Korea, Taiwan, and Japan almost simultaneously, causing PED epidemics nationwide. Therefore, the search of anti-PEDV strategies remains exceedingly actual task. Recent data demonstrated the inhibiting effect of milk small extracellular vesicles against PEDV infection in IPEC-J2 and Vero cells. Taking into the account that IPS contains milk substances, observed in our study PEDV suppression can be caused by similar anti-viral mechanisms.By supporting innate immunity and intestinal function, isotonic protein solutions (IPSs) may help in restoring the morphology and function of enterocytes after PEDV infection. To this end, the present study evaluated the effects of supplementing (or not) the drinking water of 14-days-old PEDV-infected piglets with an IPS on: the content of E-cadherin, fibronectin (two structural proteins), and interferon-alpha (IFN-α, an antiviral cytokine); the activity of metalloproteinase 9 (MMP-9, an enzyme that degrades the extracellular matrix); and the content of PEDV DNA in the rectum of piglets. The IPS-supplemented group evidenced a less abrupt decrease in E-cadherin and fibronectin and a modulation of IFN-α production and MMP-9 activity. At day 21 post infection, no PEDV DNA was detected in the rectum of piglets supplemented with the IPS. Overall, these results indicate that IPS supplementation is a viable intervention to rehabilitate the intestinal barrier integrity and function and to modulate the immune response. This is possibly done by providing amino acids that promote the metabolism of structural proteins. IPS supplementation is therefore a valid intervention to mitigate the damage inflicted by PEDV on the intestine.

Considering the results obtained here, IPS supplementation is a valid strategy to protect intestinal barrier function and ameliorate PEDV symptoms. It improves the metabolic activity of enterocytes, which enhances intercellular adhesion (E-cadherin) and ECM structure (fibronectin), reduces the MMP-induced cleaving of the ECM, and modulates IFN-α production for improved resistance to the cell damage caused by PEDV infection.