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SHORT COMMUNICATION

Pigs

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Influence of high- and low-fermentable dietary fibres in sows' diet on the colostrum potential against *Clostridioides difficile* toxin-induced effects in IPEC-J2 cells

Łukasz Grześkowiak | Wilfried Vahjen | Jürgen Zentek

Department of Veterinary Medicine, Institute of Animal Nutrition, Freie Universität Berlin, Berlin, Germany

Correspondence
Łukasz Grześkowiak, Department of Veterinary Medicine, Institute of Animal Nutrition, Freie Universität Berlin, Königin-Luise-Str. 49, 14195 Berlin, Germany.
Email: lukasz.grzeskowiak@fu-berlin.de

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Abstract

Sow colostrum has been reported to protect the IPEC-J2 cells and piglet colon tissues from detrimental effect of *Clostridioides difficile* toxins. Since dietary fibre can influence the colostrum composition in sows, we hypothesised that it can also differentially affect the colostrum potential against *C. difficile* toxin-induced effects in IPEC-J2. IPEC-J2 were incubated with colostrum from sows fed either high-fermentable sugar beet pulp (SBP) or low-fermentable lignocellulose (LNC) fibres and in combination with the toxins and analysed by trans-epithelial electrical resistance (TEER) and cell viability using propidium iodide in flow cytometry. Toxins drastically decreased the integrity of IPEC-J2. Colostrum from the sows fed either SBP or LNC exerted protective effect against toxins on IPEC-J2 integrity and this effect was numerically superior in the SBP group. Differences in the percentages of TEER between different treatments were noted after 2 h ($p = 0.043$), 3 h ($p = 0.017$) and 4 h ($p = 0.017$) of incubation and a tendency for differences was noted after 5 h of incubation ($p = 0.071$). Colostrum from either SBP- or LNC-fed sows did not protect the IPEC-J2 from toxin-induced death. Colostrum of the sows fed either high-fermentable or low-fermentable fibres has a potential to protect IPEC-J2 from the loss of integrity, which may be important in protection from *C. difficile*-infection development in neonatal piglets.

KEYWORDS
lignocellulose, piglet, sugar beet pulp, TEER, viability

1 | INTRODUCTION

Clostridioides difficile toxins are considered the primary agents causing intestinal epithelial damage being a main factor in the aetiology of *C. difficile*-infection (CDI) in neonatal piglets (Nusrat et al., 2001; Grześkowiak et al., 2018, 2020a). Specifically, toxin A (TcdA) and toxin B (TcdB) are important determinants in the veterinary diagnostic investigations of CDI (Knight et al., 2014; Grześkowiak et al., 2020b). The prevalence of toxin-positive piglets on farms and in experimental units can vary vastly and non-diarrhoeic besides diarrhoeic piglets often test positive for either TcdA or TcdB (Alvarez-Perez et al., 2009; Grześkowiak et al., 2016; Songer & Uzal, 2005). CDI among piglets are spontaneous and the reasons for that are still not known (Grześkowiak et al., 2019; Songer et al., 2000).

It has been shown that the administration of *C. difficile* toxins to intestinal porcine epithelial cells (IPEC-J2), human T84 cell line, porcine colon tissues ex vivo, or an experimental infection of piglets with toxigenic *C. difficile* types causes detrimental changes to colon epithelium, cell integrity and cell signalling, and consequently leads to intoxication and CDI development (Arruda et al., 2013; Nusrat et al., 2001; Steele et al., 2010; Grześkowiak et al., 2018, 2020a). *C. difficile*-toxin neutralising antibodies have been identified in porcine colostrum (Grześkowiak et al., 2019, 2020a). However, why some neonatal piglets are more prone to CDI and others not, remains unsolved. A lack of toxin receptors on the gut epithelium or inadequate supply of toxin-neutralising antibodies in colostrum have been proposed as possible explanations (Grześkowiak et al., 2019; Keel & Songer, 2011; Steele et al., 2012). Colostrum is rich in nutrients and bioactive compounds and is essential for the survival and health of neonatal piglets. Moreover, its composition can be influenced by the diet fed to sows during gestation and lactation. Specifically, dietary fibre has been shown to modify nutritional (increase in crude protein and biogenic amines) and immunological (reduction in lipopolysaccharide and C-reactive protein concentrations) composition of colostrum in sows with a consequence on piglet health by for instance increasing IgG-anti-lipopolysaccharide in blood serum and decreasing pre-weaning mortality (Loisel et al., 2013; Werner et al., 2014; Grześkowiak et al., 2022a).

Therefore, we hypothesised that colostrum from sows fed high-fermentable sugar beet pulp or low-fermentable lignocellulose fibres can differentially exert protective effects against *C. difficile* toxins on IPEC-J2 cells. With this in mind, we aimed to measure the trans-epithelial electrical resistance (TEER) and viability in IPEC-J2 cells exposed to sow colostrum and *C. difficile* toxins.

2 | MATERIALS AND METHODS

The animal trial was approved by the Regional Office for Health and Social Affairs (LAGeSo Reg. G0112/19). Colostrum samples were collected from 20 sows which were fed experimental gestation and lactation diets enriched with either high-fermentable sugar beet pulp (SBP; $n = 10$; inclusion rate: 15% sugar beet pulp, 3% lignocellulose) or low-fermentable lignocellulose (LNC; $n = 10$; inclusion rate: 15% lignocellulose, 3% sugar beet pulp) fibres, as previously described in detail (Grześkowiak et al., 2022b). Briefly, colostrum from randomly chosen teats was collected within 10 h after beginning of the farrowing once the afterbirth was excreted and stored frozen at −20°C until further analysis. None of the study sows were submitted to any vaccination or medication protocol before the colostrum collection. The nutrient composition and immunological traits including the presence of IgG-anti-toxin-A and -B antibodies in the collected colostrum were previously determined and published elsewhere (Grześkowiak et al., 2022a).

IPEC-J2 cells (ACC 701) were maintained as previously described (Grześkowiak et al., 2020a). The cells from passage 55 were used for the TEER and viability experiments. Briefly, the IPEC-J2 cells were seeded to a density of 5×10^5 cells/well and were maintained in the cell culture plates for 7 days at 37°C with 5% CO_2 changing to a fresh medium every second day, until they reached confluence. Thereafter, the cells were subjected to the experiments, which were performed three times and independently. Each independent experiment was performed in triplicate.

The spent culture supernatant containing *C. difficile*-toxins was prepared as previously described (Grześkowiak et al., 2020a). The confluent IPEC-J2 cells were treated with the following: (I) DMEM growth medium as a control; (II) sow individual colostrum (1–20); (III) spent culture supernatant containing toxins; and (IV) sow individual colostrum (1–20) + spent culture supernatant containing toxins. The final concentration of toxins was 16 ng/mL for TcdA and 8 ng/mL for TcdB, based on our previous data (Grześkowiak et al., 2019, 2020a). Both toxins and individual colostrum samples were applied at the same time onto the cells.

For the TEER assay, IPEC-J2 cells were transferred to ThinCertTM cell culture inserts (Greiner BioOne) and treated with colostrum and/or toxins as described above. The TEER was measured every hour until 5 h using an epithelial Voltammeter EVOM2 with a chopstick electrode STX2 (World Precision Instruments, Sarasota). The data were expressed as the percentage of TEER before incubation (Grześkowiak et al., 2020a).

To assess the impact of colostrum and toxins on cell viability, IPEC-J2 cells were incubated with colostrum samples, toxins and a combination of colostrum samples and toxins, as described above. After 2 h of incubation, the cells were washed twice in phosphate-buffered saline, trypsinisated, stained with propidium iodide and subjected to flow-cytometry analysis (MACSQuant® Analyze, Miltenyi Biotec). The data were expressed as the percentage of dead cells from the total number of cells.

All data were analysed by Kruskall–Wallis H or Mann–Whitney *U* test. Correlation analyses were assessed by Spearman's correlation analysis procedure. Differences were considered significant at $p \leq 0.05$ (SPSS v. 27).

3 | RESULTS AND DISCUSSION

Results showed that toxins decreased the integrity, as measured by TEER, of IPEC-J2 cells in a time-dependent manner. When the cells were incubated with toxins, the integrity of IPEC-J2 after 1 h of incubation decreased to 24.3% ± 3.9 of the initial TEER values before any treatment was applied to the cells. On the contrary, colostrum from sows fed either SBP or LNC exerted protective effect against toxins as indicated by TEER in IPEC-J2 cells (Figure 1). Specifically, after 2 h of incubation the TEER was 72.3% ± 1.6 in control medium, 50.3% ± 11.2 in colostrum from SBP-fed sows, 47.1% ± 6.5 in colostrum from LNC-fed sows, 8.0% ± 0.1 in toxins, 37.0% ± 17.8 in colostrum from SBP-fed sows + toxins and 18.4% ± 3.4 in colostrum from LNC-fed sows + toxins of the TEER before any treatment was applied to the cells ($p = 0.043$). Significant differences in the percentages of TEER between different treatments were noted after

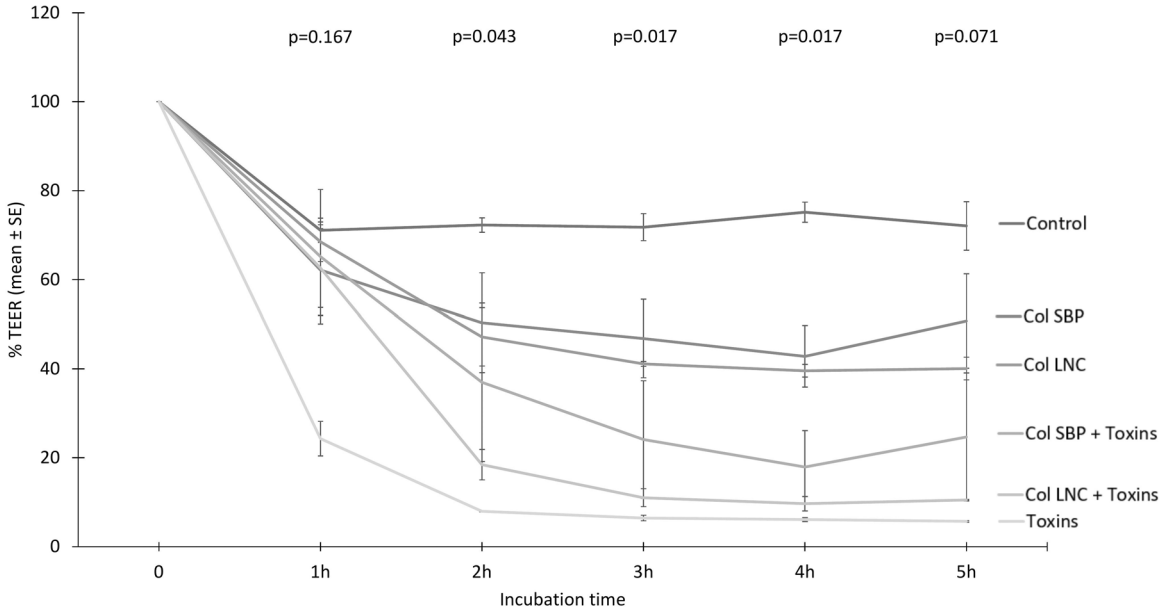

FIGURE 1 Percentage of transepithelial electrical resistance (TEER), as compared to the TEER before treatment, of IPEC-J2 cells measured in growth medium (Control), individual colostrum samples from sows fed diets containing high-fermentable sugar beet pulp (Col SBP) or low-fermentable lignocellulose (Col LNC) fibres during gestation and lactation, individual colostrum samples and toxin complex (Col SBP + Toxins, Col LNC + Toxins) or *Clostridioides difficile*-toxins alone (Toxins). The data summarises three independent experiments. The Kruskal–Wallis H test was used to test differences between the groups (significance $p \leq 0.05$).

3 h ($p = 0.017$) and 4 h ($p = 0.017$) of incubation and numerical difference was noted after 5 h ($p = 0.071$) of incubation. These results demonstrate that colostrum from the sows fed either SBP or LNC fibres during gestation and lactation exerted protective effects against *C. difficile* toxins on IPEC-J2 integrity. Specifically, colostrum from the sows fed SBP showed numerically (but not statistically) superior protection from the loss of cell integrity, as compared to colostrum from the sows fed LNC. Although the observed protection for either SBP- or LNC-sows' colostrum was time-dependent, the *in vivo* situation in which the offspring has a constant access to colostrum may be different. It has been shown that among different fibres, inclusion of SBP in the diet can influence the sow's physiology and colostrum composition, with a positive consequence on piglets' health by significantly increasing colostrum intake and decreasing pre-weaning mortality (Krogh et al., 2015; Loisel et al., 2013). Colostrum is a source of essential nutrients and immune cells, which are crucial for the piglet normal development and a resilience to diseases (Sugiharto et al., 2015; Grześkowiak et al., 2018, 2022a). Considering that *C. difficile* infections have been reported in neonatal suckling piglets, colostrum quality and its intake by the offspring seem to be crucial in prevention strategies from neonatal enteric diseases.

Previous studies demonstrate that *C. difficile* toxins target cell structures such as F-actin cytoskeleton and tight junction proteins in T84 human cell lines (Nusrat et al., 2001). We have also previously demonstrated that porcine colostrum protects the IPEC-J2 from *C. difficile* toxin-induced effects by positive influence on the cell structures such as F-actin cytoskeleton and zonula occludens-1 ZO-1 (Grześkowiak et al., 2020a). Toxin-induced cell disruption seem to be crucial for the nutrient supply and growth of *C. difficile* in the host. Recently, in IMR90 human fibroblast monolayers, *C. difficile* toxins caused notable collagen degradation leading to a release of proteins and amino acids necessary for *C. difficile* growth. Moreover, the toxin-induced inflammation influenced the potential competitors for collagen nutrients such as *Bacteroides* spp. in mice caecum (Fletcher et al., 2021). The published findings and ours demonstrate that *C. difficile* through its potent toxins is capable of successfully creating an environment suitable for its survival.

The number of dead IPEC-J2 cells was determined after 2 h of incubation with toxins, colostrum samples and its combination, by flow cytometry analysis of propidium iodide staining (Figure 2). The overall difference in the percentage of dead cells between all the treatments was significant ($p < 0.001$). The percentage of dead IPEC-J2 cells in the control medium was 5.4% ± 0.3. However, toxins significantly increased the percentage of dead cells to 13.5% ± 1.1, as compared to control ($p = 0.021$). Previously, we could demonstrate that elevated toxin concentration led to increased IPEC-J2 cell death (Grześkowiak et al., 2020a). Here, we found that cells incubated in colostrum from sows fed SBP had significantly higher percentage of dead cells (12.4% ± 1.8) than when the cells were incubated in colostrum from sows fed LNC (8.0% ± 0.6), as compared to control ($p = 0.046$ and $p = 1.000$ respectively). Moreover, colostrum from sows fed either SBP or LNC and incubated together with toxins significantly increased the percentage of dead cells, as compared to control (14.5% ± 1.8, $p = 0.005$ and 14.8% ± 2.1, $p = 0.006$ respectively). Finally, colostrum from sows fed either SBP or LNC and incubated together with toxins was not able to

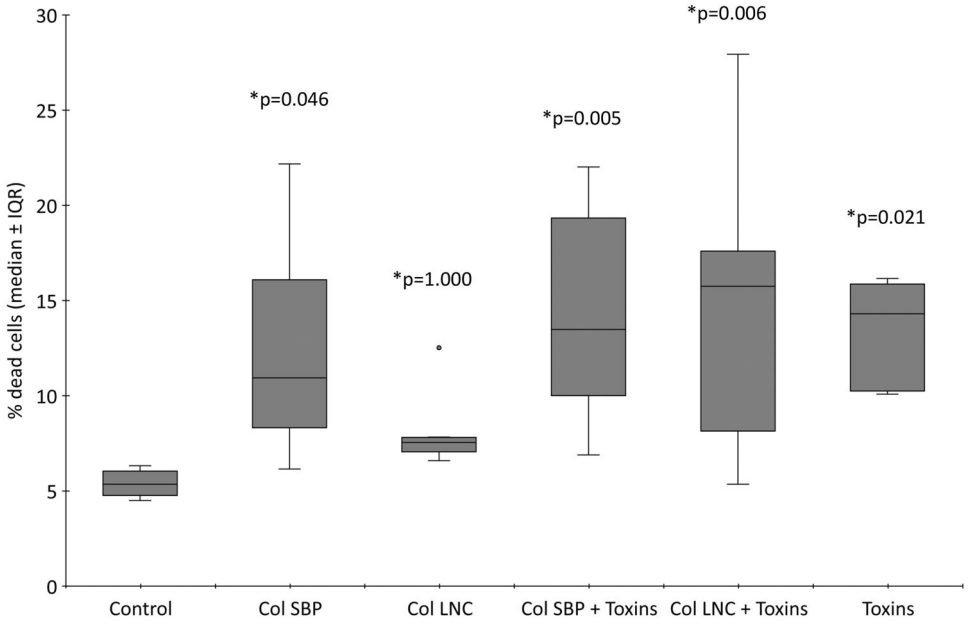

FIGURE 2 Percentage of dead IPEC-J2 cells (flow cytometry analysis of propidium iodide staining) as compared to the total number of cells, measured in growth medium (Control), individual colostrum samples from sows fed diets containing high-fermentable sugar beet pulp (Col SBP) or low-fermentable lignocellulose (Col LNC) fibres during gestation and lactation, individual colostrum samples and toxin complex (Col SBP + Toxins, Col LNC + Toxins) or *Clostridioides difficile*-toxins alone (Toxins) after 2 h of incubation. The data summarises three independent experiments. The Kruskal–Wallis H test was used to test overall difference between the groups, $p < 0.001$. A grey dot indicates an outlier. *The Mann–Whitney *U* adjusted post hoc test was used to test differences between the control and each treatment groups.

reduce the IPEC-J2 death, as compared to the cells incubated with toxins only (p = 1.000 and p = 1.000 respectively). The colostrum and milk by its nature are not sterile and therefore are populated with skin-originated microorganisms (Chen et al., 2018). The use of non-sterile colostrum in the cell assays allowed us to maintain all bioactive components and their properties. However, the presence of microorganisms in colostrum might have been detrimental to the integrity and viability of IPEC-J2 cell resulting in decreasing TEER values over time and an increase in cell death. Nevertheless, our investigations allowed us to demonstrate a protective effect of colostrum on IPEC-J2 integrity against *C. difficile* toxins in a time-dependent manner.

Interestingly, we could show that the percentage of TEER measured after 2 h of incubation inversely correlated with the percentage of dead cells (*rho* = −0.506, $p = 0.001$) independent of the treatments, indicating that with an increasing integrity the cell death was decreasing.

Different bioactive compounds present in colostrum may be responsible for the observed protective effects (Grześkowiak et al., 2019, 2022a). Previously, we have determined crude protein and crude fat and lactose content in the study colostrum samples, of which crude protein was elevated in colostrum from SBP-fed sows and lactose in colostrum from LNC-fed sows (Grześkowiak et al., 2022a). In addition, we have assessed the colostrum samples for biogenic amines, immunoglobulins and *C. difficile* toxin-neutralising IgG antibodies (Grześkowiak et al., 2022a). Here, total biogenic amines were higher in colostrum from sows fed SBP than LNC (Grześkowiak et al., 2022a). Biogenic amines have been reported to influence the intestinal cell proliferation in mice and are essential for the growth and development of the neonatal small intestine (Gómez-Gallego et al., 2017; Manjarin et al., 2014). The presence of toxin-neutralising antibodies in colostrum is an important indication of the sow's immune system function and the early immune programming in the offspring. Previous in vivo studies demonstrated protective effect of human monoclonal IgG anti-toxin antibodies against CDI development in hamsters, humans and gnotobiotic piglets (Alonso & Mahoney, 2019; Babcock et al., 2006; Cohen et al., 2014). Thus, in addition to the nourishing and growth-stimulating role of colostrum on IPEC-J2, the presence of toxin-neutralising IgG antibodies may have contributed to the observed effects on IPEC-J2 integrity.

Overall, feeding the sows either high-fermentable SBP or low-fermentable LNC fibres during gestation and lactation equally protects the IPEC-J2 cell integrity against *C. difficile* toxins but high-fermentable SBP is numerically superior to low-fermentable LNC fibres. This may have important biological significance and implications in prevention from the development of *C. difficile*-infection in neonatal piglets by means of dietary interventions in gestating and lactating sows.

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CONFLICTS OF INTEREST STATEMENT

The authors declare no conflicts of interest.

DATA AVAILABILITY STATEMENT

None of the data was deposited in an official repository. The data that support the study findings are available to reviewers, or available from the authors upon request.

ORCID

Łukasz Grześkowiak http://orcid.org/0000-0002-0831-4077

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