

Effects of Deoxynivalenol on General Performance and Electrophysiological Properties of Intestinal Mucosa of Broiler Chickens

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ABSTRACT A feeding trial was conducted to evaluate the effects of diets contaminated with deoxynivalenol (DON) on the performance of broilers and on the electrophysiological parameters of the gut. The control group was fed the starter and finisher diets without addition of DON. Another group of broilers was fed the starter and finisher diets with 10 mg/kg DON, whereas another group was fed the DON-contaminated diets supplemented with a microbial feed additive (*Eubacterium* sp.). The diets were provided ad libitum for 6 wk. DON had no effect ($P > 0.05$) on feed consumption, feed conversion, or body weight. The effect of DON on the electrophysiological parameters of the jejunum was studied in vitro using isolated gut mucosa in Ussing chambers. At the end of the feeding period, 7 birds from each group were killed, and the basal and glucose stimulated transmural potential difference (PD), short-circuit current (Isc), and electrical resistance (R) were measured in the isolated gut

mucosa to characterize the electrical properties of the gut. The transmural PD did not differ ($P > 0.05$) among groups. The tissue resistance was greater ($P < 0.05$) in birds receiving DON and the microbial feed additive than in the controls and DON group. Addition of D-glucose on the luminal side of the isolated mucosa increased ($P < 0.05$) Isc in the control and DON-probiotic (*Eubacterium* sp.; PB) groups, whereas it decreased ($P < 0.05$) in the DON group indicating that the glucose-induced Isc was altered by DON. Addition of the eubacteria to the DON contaminated feed of the broilers led to electrophysiological properties in the gut that were comparable with those of the control group. It could be concluded that 10 mg/kg DON in the diet impaired the Na⁺-D-glucose cotransport in the jejunum of broilers. In the absence of clinical signs, and without impaired performance, DON appeared to alter the gut function of broilers. The addition of *Eubacterium* sp. may be useful in counteracting the toxic effects of DON on intestinal glucose transport.

(Key words: chicken, deoxynivalenol, microbial feed additive, glucose absorption, electrophysiological parameter)

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INTRODUCTION

One of the most important mycotoxins in feedstuffs for poultry in Austria and other European countries is deoxynivalenol (DON). DON is a secondary metabolite produced by *Fusarium graminearum* or *Fusarium culmorum* mainly in corn and wheat during the maturing and harvesting stages (Cote et al., 1985). High concentrations can be expected under cold and wet growing conditions, and DON is the most prevalent trichothecene in crops used for food and feed production (World Health Organization, 1993). More relevant to livestock production is that moderate to low levels of this toxin can cause a number of effects associated with poor performance and altered immunity (Pier et al., 1980; Swamy et al., 2004).

Feeds contaminated with mycotoxins pose a health risk to animals and, as a consequence, may cause economic losses, a result of the lower efficacy of livestock production. Furthermore, either directly or indirectly (carry over to animal products), contaminated feedstuffs may also pose a health risk to humans.

Poultry are less sensitive to DON than other species (Böhm, 2000; Razzazi-Fazeli et al., 2003). Dänicke et al. (2001) reviewed the literature regarding the effects of DON on the performance of broilers and concluded that dietary concentrations greater than 5 ppm are necessary to cause detrimental effects. Even higher concentrations did not consistently induce detrimental effects, and growth-promoting effects were observed especially at moderately high concentrations of DON. A review of the literature indicates that dietary concentrations of DON

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Abbreviation Key: DON = deoxynivalenol; Isc = short-circuit current; PB = probiotic (*Eubacterium* sp.); PD = transmural potential difference; Rt = electrical resistance of the tissue.

below 15 mg/kg had no adverse effect ($P > 0.05$) on BW gain, feed consumption, or feed efficiency of broilers (Hulan and Proudfoot, 1982; Bergsjö and Kaldhusdal, 1994; Harvey et al., 1997; Kubena et al., 1997; Leitgeb et al., 1999, 2000). An increase ($P < 0.05$) in feed efficiency has been observed in broilers fed a diet with 16 ppm DON (Huff et al., 1986; Kubena et al., 1989a). Feed refusal and reduced weight gain are found when the dietary concentration of DON reaches 16 to 20 ppm (Kubena et al., 1987, 1988, 1989b; Kubena and Harvey, 1988; Harvey et al., 1991). Dänicke et al. (2003) have recently published effects of different concentrations of *Fusarium* toxins (major toxin was DON) on chickens. The authors observed significantly decreased feed intake with increasing proportions of contaminated wheat and significantly decreased live weight with 10 and 14 mg of DON/kg of feed. Therefore, it is not possible to establish a simple dose-response relationship between growth depression and dietary concentrations of DON for broilers, unlike for pigs. Small erosions of the gizzard mucosa have been observed in birds fed a diet containing 82.8 mg DON/kg for 27 d (Lun et al., 1986). Increased absolute and relative gizzard weights and lesions of the proventriculus are interpreted by these authors to be a consequence of an irritation of the upper gastrointestinal tract. Hematological parameters, serum minerals, and glucose levels decrease ($P < 0.05$) when up to 18 mg of DON/kg were fed (Kubena et al., 1985). Detoxification of DON is of major practical interest, and microbial degradation might be a possible method for accomplishing it (Binder et al., 1998). DON has been reported to be completely transformed to de-epoxy-DON after incubating DON for 96 h with the contents from large intestines of hens (He et al., 1992). Furthermore, Fuchs et al. (2002) found that a strain of *Eubacterium* sp. (DSM 11798) can degrade deoxynivalenol, T-2 toxin, HT-2, scirpenol, and diacetoxyscirpenol.

There is a dearth of information available regarding the effects of DON on gut physiology and the electrical properties of the intestine of the chicken. Grubb et al. (1987) have reported that the avian intestine has a regional electrical profile different from that of mammals. In the chicken, the region with the highest electrical resistance is the duodenum, whereas in the rat and the rabbit the region of greatest resistance is the colon (Powell, 1987). In the study reported here, preparations of the jejunum mucosa were used to describe the electrical phenomena occurring in the epithelium. Transmural potential difference (transepithelial electrical potential; PD) constitutes an important electrophysiological parameter, which reflects the functional state of a tissue. PD is induced as a result of the absorption of sodium and the secretion of chloride ions (Wright, 1983; Boucher, 1994).

Amat et al. (1999), using male White Leghorn chickens, reported that addition of 5 mmol/L D-glucose on the mucosal side produced a 20% increase in short-circuit

current (Isc) in different parts of small and large intestines relative to basal values. This increase was higher ($P < 0.01$) in the rectum than in the rest of the intestine. Scharer (1972) and Lerner et al. (1976), in studies comparing the jejunum and the ileum of chicken, demonstrated that the former was characterized by relatively higher transport rates of several nutrients. Studies comparing the jejunum and the proximal cecum have indicated that the jejunum has a higher total transport capacity for sugar than the proximal cecum (Moreto et al., 1991). Maximal transport capacity values for methyl-D-glucoside showed that the jejunum is the segment that is best suited for Na⁺-mediated uptake (Ferrer et al., 1986).

Ten parts per million of DON reduced ($P < 0.05$) intestinal absorption of D-glucose in mice (Hunder et al., 1991). Dose-dependent studies of the effects of DON at 48 h, on sugar uptake by human intestinal epithelial cells, indicated that DON inhibited uptake of α -methyl-glucose, resulting in 50% decrease at 10 μ mol/L ($P < 0.05$) and a maximal effect at 100 μ mol/L ($76 \pm 1.6\%$ of inhibition; $P < 0.01$). DON selectively modulated the activities of intestinal transporters. The D-glucose/D-galactose sodium-dependent transporter (SGLT1) was strongly inhibited by DON (50% inhibition at 10 μ mol/L). On the other hand, passive transporters of D-glucose were only slightly inhibited by DON (15% inhibition at 1 μ mol/L; Maresca et al., 2002).

There are no reports in the literature to date regarding the impact of DON on electrophysiological properties of intestinal mucosa of broiler chickens. Such data would add to the understanding of the mode of action of DON on gut physiology and provide new aspects to the definition of maximum tolerable DON levels in feedstuffs. The objective of the present study was to investigate the influence of 10 mg/kg DON in the presence or absence of a microbial feed additive on the performance of broiler chicks and the electrical properties of isolated jejunum mucosa. The electrogenic effect of adding D-glucose on the intestinal mucosal side as an indicator of nutrient-stimulated Na⁺ absorption across apical Na⁺-D-glucose cotransporter was also investigated.

MATERIALS AND METHODS

Birds and Housing

Two hundred seventy-seven, 1-d-old broiler chicks (males and females) were obtained from a commercial hatchery.² The birds were weighed at the beginning of the experiment, randomly divided without regard to sex into 3 groups, and housed in pens of identical size (3.50 \times 3.25 \times 3 m) in a deep litter system. Wood shavings were used as the litter material.

Treatment and Diets

The control group (n = 91) was fed starter and grower diets based on wheat, soybean meal 48, maize, rapeseed oil, and a premix with vitamins, minerals, amino acids,

²Ross E032, Geflügelhof Schulz, Schulzweg 16, 8301 Lassnitzhöhe, Austria.

TABLE 1. Composition of the experimental diet¹ (%)

Ingredient	Starter	Grower
Wheat	39.25	38.00
Soybean meal 48	29.00	27.00
Maize	20.00	22.65
Rape seed oil	6.25	7.00
Dicalcium-phosphate	0.58	0.70
Lys	0.26	0.15
Met	0.28	0.25
Premix ²	4.38	4.25
Calculated composition		
Dry matter	88.06	88.15
Crude protein	21.13	20.13
ME (MJ/kg)	12.69	12.95
Crude fiber	2.41	2.37
Crude fat	8.21	9.01
Lys	1.34	1.19
Met	0.80	0.75
Ca	1.01	1.00
P	0.71	0.71
Na	0.12	0.12
Mg	0.14	0.14

¹Ten milligrams of deoxynivalenol (DON)/kg was added to starter and grower diets to constitute the DON group. Ten milligrams of DON/kg and 2.5×10^8 cfu/kg *Eubacterium* sp. DSM 11798 (PB) was added to constitute the DON-PB group.

²Produced by MIAVIT GmbH & Co., KG, Essen, Germany. Composition per kilogram: 185 g of calcium, 58 g of phosphorus, 50 g of methionine, 25 g of sodium, 250,000 IU of vitamin A, 82,500 IU of vitamin D₃, 826 mg of vitamin E, 50 mg of vitamin B₁, 225 mg of vitamin B₂, 75 mg of vitamin B₆, 825 mg of vitamin B₁₂, 37 mg of vitamin K₃, 12,500 mg of cholinchloride, 1,000 mg of nicotinic acid, 245 mg of calcium-pantothonate, 25 mg of folic acid, 1,240 mg of biotin, 1,500 mg of iron, 500 mg of copper, 1,750 mg of manganese, 1,250 mg of zinc, 32 mg of iodine, 5 mg of cobalt, 6 mg of selenium.

salt, and dicalcium phosphate. The DON group (n = 95) was fed the starter and grower diets supplemented with 10 mg/kg DON.³ The DON-probiotic group (n = 91) was fed the same starter and grower diets supplemented with 10 mg/kg DON and 2.5×10^8 cfu/kg *Eubacterium* sp. DSM 11798 (PB; probiotic).⁴ Chicks were fed the starter diets from d 1 to 13 and the grower diets from d 14 to 42 (Tables 1 and 2). Feed and water were offered ad libitum. Chicks were kept at 35°C for the first week of life, and then the temperature was gradually decreased 2°C weekly to 25°C for the remainder of the experiment. During the first 2 wk, chicks were provided with 24 h of light, after which lighting was decreased gradually (2 h daily) to 20 h by the third week and remained there until the end of the trial. Representative feed samples were taken at the beginning of the starter and grower periods and were analyzed for nutrient content and *Fusarium* mycotoxins.

Traits

Feed consumption, weight gain, and feed conversion were measured weekly. Deoxynivalenol and the other

³Provided by Biomim, GTI GmbH, Herzogenburg, Austria.

⁴A product of Biomim.

⁵A product of Sigma-Aldrich Chemie GmbH, Schnellendorf, Germany.

⁶Ø 63 pH meter, Beckman Coulter GmbH, Europark Fichtenhain B13, Krefeld, Germany.

TABLE 2. Analyzed composition of the experimental diet (% in dry matter)

Ingredient	Starter	Grower
Dry matter	91.00	91.40
Crude protein	25.51	25.09
Crude fiber	2.30	2.29
Crude fat	9.00	10.10
Crude ash	6.30	6.02
Starch	49.01	52.74
Sugar	2.55	2.52
Lys	1.24	1.09
Met	0.86	0.84
Ca	1.29	1.24
P	0.89	0.92
Na	0.38	0.36
Mg	0.23	0.20
K	1.04	1.05
Cu	0.003	0.003
Zn	0.02	0.01
Fe	0.05	0.03
Mn	0.02	0.02

trichothecenes were determined in the diets (Table 3) by HPLC according to the method of Valenta et al. (2002). Seven birds from each group were kept after the growth trial (42 d) for the in vitro experiments. Experiments with isolated jejunum mucosa were carried out using 6- to 8-wk-old chickens, which represents the period in which monosaccharide transport (Soriano and Planas, 1998) and electrical variables do not change appreciably and are similar for males and females.

Tissue Preparation

Twenty-one birds (7 per group, 6 jejunum segments/bird) were killed by cervical dislocation. For preparation of isolated jejunal segments, the gastrointestinal tract was removed within 3 min after exsanguination. An intestinal segment was immediately taken from proximal jejunum. Manipulation and experimental procedures were performed in accordance with Amat et al. (1999). The intestine was rinsed with ice-cold Ringer buffer and transported in ice-cold oxygenated incubation buffer to the laboratory. The intestinal segment was opened along the mesenteric border and washed free of intestinal contents with Ringer solution at 4°C.

Tissues were placed in the same cold Ringer buffer until they were mounted in the Ussing chamber. After preparation of unstripped intestinal sheets, the tissue was mounted in modified Ussing chambers with an active area of 1 cm² (Naftalin and Holman, 1974). The serosal and mucosal surfaces of the tissues were bathed in 5 mL of Ringer solution with the following composition (mmol/L): CaCl₂, 1.2; MgCl₂, 1.2; Na₂HPO₄, 2.4; NaH₂PO₄, 0.4; NaHCO₃, 25; KCL, 5; NaCl, 115; mannitol, 20 for the serosal side. Ringer solution was added to mucosal side and 5 mmol D-glucose was added instead of mannitol. All chemicals⁵ were dissolved in distilled water and mixed thoroughly in a 1-L flask. The pH of the solution was adjusted to 7.4 using a pH meter.⁶ The incubation medium was continuously gassed with a mix-

TABLE 3. Mycotoxin composition ($\mu\text{g}/\text{kg}$) of contaminated and noncontaminated feed mixture

	Diet					
	Control		DON		DON-PB	
	Starter	Grower	Starter	Grower	Starter	Grower
DON ¹	408	444	9,511	9,156	8,356	9,444
Acetyldeoxynivalenol	ND ²	ND	ND	ND	ND	ND
Nivalenol	ND	ND	ND	ND	ND	ND
Fusarenon X	ND	ND	ND	ND	ND	ND

¹Deoxynivalenol.

²Not detected ($<50 \mu\text{g}/\text{kg}$).

ture of 95% O₂ and 5% CO₂, and the temperature of the mixture was kept at 38°C. Continuous oxygenation provided recirculation of the incubation solutions by means of a gas lift.

Transmural potential difference (PD), I_{sc}, and electrical resistance (R_t) were measured with a microprocessor-based voltage-clamp device.⁷ Up to 6 chambers were used for each bird. The technique is based on the original procedures to determine electrical variables (Ussing and Zerahn, 1951; Parsons, 1968). The spontaneous PD was measured between calomel electrodes connected to the chamber by 3 mol/L KCl agar bridges. The possible PD existing between the calomel cells was automatically compensated at the beginning of the experiment when the tissue was not in place. Except for short periods, the PD was measured with an open-circuit, and the tissue was short-circuited by passing the appropriate current through the Ag/AgCl electrodes. The transmural electrical resistance of the mucosa was determined automatically by periodically clamping the transmembrane potential to a fixed voltage (0 mV) and measuring the current required to accomplish this. The solution resistance was determined before the membrane was in place; the resistance was measured by the voltage deflection caused by a current pulse across a chamber containing the bathing solution. Five milliliters of Ringer solution was presented on the mucosal and serosal sides.

The basal measurements of PD, I_{sc}, and R_t were taken after a stabilization period of 30 min. When the tissue was stabilized, the 5 mL of Ringer solution on the luminal side was replaced by 5 mL of glucose buffer. The electrical response to glucose was measured as the peak response obtained approximately 1 min after the addition of the substrate. The basal PD, I_{sc}, and R_t are expressed as actual values, whereas the effect of D-glucose to the mucosal side on the electrical variables is shown as the amount of change.

Statistical Analysis

Statistic SPSS⁸ program version 10.0 was used for data analysis. Kolmogorov Smirnov test was used to test the

normal distribution of the data. Results are given as means \pm SEM. Body weight and feed conversion data were compared between groups by one-way ANOVA, and, subsequently, Duncan's multiple range test was used for group comparison. Basal values of PD, I_{sc}, and R_t (6 replicates per bird) were compared by ANOVA between groups, and I_{sc} was compared within groups (replicates) by ANOVA. Paired samples *t*-test was used for the comparison of the different measurements within the same group. Significance was denoted by $P < 0.05$.

RESULTS

Growth Trial

All birds appeared clinically normal during the entire feeding trial. The general performance (weight gain, feed efficiency) of the birds was not influenced ($P > 0.05$) by feeding DON (Tables 4 and 5). Feed intake was not influenced ($P > 0.05$) by the presence of DON in the diet, and indeed a slight increase in feed intake was observed during the experiment for this group compared with the control group. Feed conversion rate was slightly improved for the DON fed birds compared with the control group during the starter period, but a slight numerical decrease of feed conversion was noted for the finishing period. Addition of PB to the 10 mg/kg DON diet did not improve the general performance of broilers. However, the mean BW and the mean feed intake over the course of the experiment was slightly higher for broilers fed the DON containing diet supplemented with PB compared with the control or DON fed birds.

In Vitro Studies

The basal values for PD and R_t are shown in Table 6. For the control group, the DON group, and the DON-PB group, the PD's were ($P > 0.05$) -7 ± 3.22 mV, -12 ± 3.32 mV and -7 ± 3.00 mV, respectively. The tissue resistance was higher ($P < 0.05$) ($501 \pm 28,55 \Omega \cdot \text{cm}^2$) for DON-PB fed birds compared with the control birds ($380 \pm 26.73 \Omega \cdot \text{cm}^2$), and it was numerically higher for the DON group ($406 \pm 14 \Omega \cdot \text{cm}^2$) compared with the controls.

Addition of 5 mmol/L D-glucose on the luminal side of the jejunum increased ($P < 0.05$) I_{sc} in the control and DON-PB fed groups, reflecting a higher net flux of sodium

⁷Mussler, Microclamp, Aachen, Germany.

⁸SPSS GmbH, SPSS Inc., Munich, Germany.

TABLE 4. Body weights (g) of the experimental birds¹

Age	Control (n = 91)	DON (n = 95)	DON-PB (n = 91)	P
Day 1	35 ^b ± 0.51	41 ^a ± 0.52	38 ^c ± 0.49	0.001
Week 1	87 ^b ± 1.9	98 ^a ± 1.8	89 ^b ± 2.1	0.001
Week 2	279 ^b ± 6.0	298 ^a ± 5.6	291 ^{ab} ± 6.6	0.052
Week 3	627 ± 9.6	644 ± 10.8	627 ± 11.2	0.399
Week 4	1,138 ± 14.9	1,154 ± 17.2	1,134 ± 17.7	0.687
Week 5	1,768 ± 22.9	1,779 ± 27.4	1,784 ± 23.7	0.784
Week 6	2,351 ± 31.4	2,399 ± 34.3	2,425 ± 30.9	0.263

^{a-c}Within the same row means with different letters are significantly different ($P < 0.05$; Duncan's test). Results are reported as means ± SEM.

¹DON = deoxynivalenol; PB = probiotic (*Eubacterium* sp.).

from the mucosal to the serosal side compared with basal conditions, whereas this parameter decreased ($P < 0.05$) for the group fed DON (Table 7). The effects of Isc in the control and DON-PB birds were similar with increases of 34 and 32 $\mu\text{A}/\text{cm}^2$, respectively. This is equivalent to an increase of about 2 times that for the basal values. For the DON group, the Isc decreased by 7 $\mu\text{A}/\text{cm}^2$ relative to the basal values.

DISCUSSION

The current study demonstrated clear effects of DON on intestinal physiology of broilers even in the absence of clinical signs or impaired growth. To our knowledge, this is the first report demonstrating the effect of 10 mg/kg purified DON on the electrophysiological properties of intestinal mucosa of broilers.

Growth Performance

Body weight, body weight gain, and feed conversion were not adversely affected ($P > 0.05$) by inclusion of 10 mg/kg DON in broiler diets. In fact, at wk 1 and 2 of the experiment, the chicks receiving the DON diet were heavier ($P > 0.05$) than chicks fed the control diet. These findings are in agreement with those of Hamilton et al. (1985, 1986), who indicated that chicks fed diets with 4.6 mg/kg DON grew faster than chicks on a control diet. Our results from 3 wk to the end of the experiment showed that there was no adverse effect on performance of broilers. This result confirms the findings of Hulan and Proudfoot (1982), who reported no effects on body weight

gain or feed efficiencies when naturally contaminated wheat-based diets containing up to 1.9 mg of DON/kg were fed to broilers for 28 d. Furthermore, Bergsjø and Kaldhusdal (1994) reported that the general performance of broilers was not affected when fed oats which were naturally contaminated with deoxynivalenol (3.4 mg of DON/kg of feed). Swamy et al. (2002) observed that feeding broilers grains that were naturally contaminated with DON (9.7 mg/kg DON) did not affect feed consumption, weight gain, or feed efficiency. Furthermore, Kubena et al. (1997), Harvey et al. (1997), and Li et al. (2003) have indicated that concentrations of dietary DON below 16 mg/kg had no adverse effect on BW gain, feed consumption, or feed gain of broilers. Moran et al. (1982) reported that growth and feed intake of broiler chickens from 6 to 11 d of age were affected ($P > 0.05$) but not until the DON content of the artificially contaminated diet exceeded 116 mg of DON/kg.

The concentration of DON in the present study fell in a range used by the other researchers. However, in general, artificially contaminated diets with purified DON are less toxic than naturally contaminated diets (Canady et al., 2002). Furthermore, a median lethal dose (LD₅₀) of 140 mg of DON/kg of body weight (oral dose) in 1-d-old broiler chicks indicates the relative insensitivity of broilers to DON toxicity (Huff et al., 1981).

Electrophysiological Parameters

The Ussing chamber was initially used in studies of electrophysiological parameters of isolated frog skin (Koefoed-Johnsen and Ussing, 1958). At present this ap-

TABLE 5. Feed intake and feed conversion rates of the experimental birds

Age	Feed intake (g/bird per week)			Feed conversion rates		
	Control (n = 91)	DON ¹ (n = 95)	DON-PB (n = 91)	Control (n = 91)	DON (n = 95)	DON-PB (n = 91)
Week 1	96	108	93	1.88	1.89	1.82
Week 2	367	347	367	1.91	1.73	1.83
Week 3	453	473	522	1.31	1.38	1.55
Week 4	878	854	842	1.71	1.68	1.63
Week 5	985	1,010	1,000	1.57	1.62	1.54
Week 6	1,075	1,152	1,200	1.84	1.95	1.87

¹DON = deoxynivalenol; PB = probiotic (*Eubacterium* sp.).

TABLE 6. The transmural potential difference and electrical tissue resistance across the isolated mucosa of broilers

Item	Control (n = 7)	DON ¹ (n = 7)	DON-PB (n = 7)	P
Transmural potential difference (mV)	-7 ± 3.2	-12 ± 3.3	-7 ± 2.9	0.422
Electrical tissue resistance (Ohm·cm ²)	380 ^b ± 26.7	406 ^b ± 13.5	501 ^a ± 28.6	0.002

^{a,b}Within the same row means with different letter are significantly different ($P < 0.05$, Duncan's test). Results are reported as means ± SEM.

¹DON = deoxynivalenol; PB = probiotic (*Eubacterium* sp.).

paratus is commonly used for electrophysiological studies of all epithelial tissues (Tyrakowski et al., 1998a; Kosik-Bogacka et al., 2002).

The small and large intestines of birds have high absorptive rates for water and electrolytes. The movements of ions responsible for the electrical current across the epithelium are mainly result of absorption of Na⁺ and secretion of Cl⁻ (Skadhauge, 1981; Grubb, 1991). The current induced by ion transport was recorded as changes in PD.

In this study, we measured the PD, Rt, and Isc of all groups using the Ussing chambers. The PD was reduced in broilers that were fed the diet containing 10 mg/kg DON. This response could be attributed to the inhibitory effect of DON on Na⁺-transport, a result of the effect of DON on sodium cotransporter (Maresca et al., 2002). This is similar to the effect of amiloride, an inhibitor of sodium ion transport (Renard et al., 1994; Tyrakowski et al., 1998b; Kosik-Bogacka et al., 2003), which decreases PD.

A profile of tissue resistance obtained from jejunum indicated that the broilers fed the DON-PB diets had greater ($P < 0.05$) resistance than the other groups. Furthermore, the resistance was numerically higher in the DON group compared with controls. This finding may be ascribed to a relatively tighter epithelium (Amat et al., 1996). The PB may have produced a protective barrier by causing increased thickening of the mucosal layer (Simmering and Blaut, 2001); however, this was not verified in the present study.

Glucose, like other nutrients, is transported by carrier systems, and they are usually cotransported with Na⁺. Thus, if those nutrients are added to the mucosal side of intestinal tissues, the carrier-mediated transport is stimulated, and there is a concomitant rise in the uptake of Na⁺. Stewart and Turnberg (1987) measured a depolarization of 7 mV when a rat ileum was incubated in the presence of 10 mmol/L D-glucose. The depolarization of the brush border membrane and the increase in the Na⁺

cytosolic concentration stimulated the Na⁺-K⁺-adenosine triphosphatase, which, in turn, increased the net flux of Na⁺ from the mucosal side to the serosal side. All of these processes modified the electrical variables of the tissue such as PD and Isc. The Na⁺-coupled glucose uptake across sodium glucose-linked transporter-1 is present in all regions of the small and large intestines of chickens (Obst and Diamond, 1989; Ferrer et al., 1994). Maximal transport capacity values for D-glucose has indicated that the jejunum is the segment that is most suited for Na⁺-mediated uptake (Amat et al., 1996).

Lind et al. (1980), Tsuji et al. (1985), Shimada and Hoshi (1986), Luppa et al. (1987), and Moreto et al. (1998) were all able to correlate the carrier-mediated glucose transport system and Isc. In the present study, addition of 5 mmol of glucose to the mucosal side increased the Isc ($P < 0.05$). This finding supports the results of Amat et al. (1999) and Aschenbach et al. (2002), who found that the increase in Isc after glucose addition to luminal side was due to a stimulation of transepithelial Na⁺ transport.

Deoxynivalenol seems to have specific effects by reducing nutrient absorption (Rotter et al., 1996). DON impaired the intestinal transfer and uptake of nutrients such as D-glucose (Hunder et al., 1991), which seemed to occur at the gut level without negative impact on performance. In the present study, DON decreased ($P < 0.05$) Isc after addition of D-glucose. These results confirm that DON decreased the absorption of glucose. This result could be attributed to the effect of DON on D-glucose/sodium-dependent transporter (Maresca et al., 2002).

According to results of experiments to date, it appears that microorganisms are the main living organisms applicable for mycotoxin biodegradation. Further screening of microorganisms may lead to the detection of more efficient and better applicable bacteria. A review of the literature revealed that DON is degraded by *Eubacterium* sp. DSM 11798, which transforms DON into its metabolite [DOM-1 the nontoxic de-epoxide of DON (Binder

TABLE 7. The short-circuit current (Isc) in isolated mucosa of broilers after addition of D-glucose

Treatments	Control	DON ¹	DON-PB	P
Basal Isc (μA/cm ²)	16 ± 7.56	33 ± 7.75	18 ± 7.15	0.216
Isc 2 min after addition of glucose (μA/cm ²)	50 ^a ± 7.65	26 ^b ± 7.49	50 ^a ± 7.65	0.046
Isc 30 min after glucose addition (μA/cm ²)	18 ± 6.73	23 ± 6.48	33 ± 7.30	0.315

^{a,b}Within the same row means with no common superscript differ statistically ($P < 0.05$, Duncan's test). Results are reported as means ± SEM.

¹DON = deoxynivalenol; PB = probiotic (*Eubacterium* sp.).

et al., 1997, 1998)]. Fuchs et al. (2002) also reported that the *Eubacterium* sp. is capable of detoxifying DON. The results reported here indicate that PB can influence electrophysiological parameters of the gut. This result may be due to improved nutrient absorption because of a reduction of toxin resulting from the biotransformation of DON to de-epoxy-DON, resulting in a loss of toxicity (Fuchs, 1999).

In conclusion, feeding diets contaminated with DON diets at 10 mg/kg had no effect on the growth performance of broilers. However, DON appears to have a specific effect by reducing nutrient absorption. Indeed this study revealed that DON impaired the intestinal transfer and uptake of nutrients such as D-glucose. This seems to occur at the gut level; however, it did not negatively impact growth performance. DON alters gut function in broilers. Ten parts per million of DON decreased ($P < 0.05$) Isc after addition of glucose to the luminal side of jejunum, which could indicate that the glucose induced Isc was altered by DON. Furthermore, the supplementation of PB (*Eubacterium* sp.) was useful in counteracting toxic effects of DON on intestinal sodium and glucose transport.

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