



Research article

Effects of feeding corn naturally contaminated with *Fusarium* mycotoxins and/or a modified yeast cell wall extract on the performance, immunity and carcass characteristics of grain-fed veal calves

L.M. Martin^a, K.M. Wood^a, P.L. McEwen^b, T.K. Smith^a, I.B. Mandell^a,
A. Yannikouris^c, K.C. Swanson^{a,*}

^a Department of Animal and Poultry Science, University of Guelph, 491 Gordon St., Guelph, ON, Canada N1G 2W1

^b University of Guelph, Ridgetown Campus, Ridgetown, ON, Canada N0P 2C0

^c Alltech Inc., Center for Animal Nutrigenomics and Applied Animal Nutrition, Nicholasville, KY 40356, USA

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ABSTRACT

Thirty-two grain-fed veal calves (177 ± 6.7 kg body weight (BW)) were used in a completely randomized block design experiment to determine effects of feeding corn diets naturally contaminated with *Fusarium* mycotoxins and/or a modified yeast cell wall extract (YCW) on performance, immunity and carcass characteristics. Calves were fed one of four dietary treatments in individual pens for at least 84 days either: (1) control corn + supplemental pellet (CC), (2) control corn + YCW supplemental pellet (CY), (3) mycotoxin contaminated corn + supplemental pellet (MC), and (4) mycotoxin contaminated corn + YCW supplemental pellet (MY). Diets consisted of 750 g/kg whole corn grain and 250 g/kg supplemental pellet with corn as the source of food-borne mycotoxins. The major contaminants present in the contaminated diets (average concentrations of MC and MY) were deoxynivalenol (DON; 10.27 mg/kg), 15-acetyl DON (1.27 mg/kg) and zearalenone (1.84 mg/kg). Final BW and total BW gain of the calves were not different between treatment groups. However, ADG tended ($P=0.07$) to be higher and F:G was decreased ($P=0.003$) in calves fed contaminated diets. Haptoglobin, fibrinogen and IgA concentrations did not differ between treatments. Concentrations of IgG were lower ($P=0.003$) in calves receiving YCW. Plasma urea N and glucose concentrations were increased ($P<0.001$) and decreased ($P=0.004$), respectively, in calves fed contaminated diets. The weights (g and g/kg BW) of the liver, kidney, heart and lungs did not differ between treatments. Hot carcass weight, dressing yield, back fat, longissimus muscle area and colour Minolta L^* value were also not different between treatments. These data indicate that veal calves are able to tolerate a moderate feeding level of corn grains naturally contaminated with *Fusarium* mycotoxins and that small improvements in F:G occurred. As there were generally no negative effects of dietary mycotoxin on performance, the efficacy of YCW as a mycotoxin adsorbent could not be assessed, but YCW alone had minimal effects on performance.

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Abbreviations: ADF, acid detergent fibre; ADG, average daily gain; aNDF, neutral detergent fibre; BF, back fat; BW, body weight; CC, control corn + control pellet; CY, control corn + modified yeast cell wall extract pellet; CP, crude protein; DM, dry matter; DON, deoxynivalenol; F:G, feed:gain; HCW, hot carcass weight; IgA, immunoglobulin A; IgG, immunoglobulin G; LMA, longissimus muscle area; MC, mycotoxin contaminated corn + control pellet; MY, mycotoxin contaminated corn + modified yeast cell wall extract pellet; PUN, plasma urea N; YCW, modified yeast cell wall extract; ZEN, zearalenone.

* Corresponding author. Tel.: +1 519 824 4120x56627; fax: +1 519 836 9873.

E-mail address: kswanson@uoguelph.ca (K.C. Swanson).

1. Introduction

The *Fusarium* mycotoxins encompass a wide range of compounds with varying structures (Santin, 2005; D'Mello and Macdonald, 1997) and can be found throughout much of the world with some compounds naturally occurring at relatively high concentrations (Binder et al., 2007). Mycotoxin contamination of grains, such as wheat, corn and barley, has been recognized as an important issue in animal production systems as *Fusarium* mycotoxins can impact growth, reproduction and immune status of animals (Morgavi and Riley, 2007).

The rumen environment has the capability to detoxify *Fusarium* mycotoxins to varying degrees and therefore ruminants are considered to be less sensitive to these compounds than other production livestock species (Dänicke et al., 2005). However, studies have indicated that milk composition, weight gain, immune function and reproductive performance can be affected in ruminants consuming a contaminated feed (Charmley et al., 1993; Noller and Stob, 1979; Buening et al., 1982; Jouany and Diaz, 2005). Effects of *Fusarium* mycotoxins on grain-fed veal calves have not been previously studied, even though these animals are fed high proportions of susceptible grains. Veal calves also may not have fully developed rumens and therefore may be partly lacking the protection that a fully functional rumen can provide against mycotoxins.

Feed additives, such as modified yeast cell wall extract (YCW), have been shown to alleviate some effects of mycotoxins in swine (Swamy et al., 2003), poultry (Chowdhury et al., 2005a,b), horses (Raymond et al., 2003) and dairy cattle (Korosteleva et al., 2007) by binding of mycotoxins to make them unavailable in the gastrointestinal tract.

The aim of the current study was to determine effects of feeding a diet naturally contaminated with *Fusarium* mycotoxins and/or YCW on performance, immunity and carcass characteristics of grain-fed veal calves.

2. Materials and methods

2.1. Experimental animals and diets

Holstein bull calves ($n=32$; mean initial body weight (BW) \pm SEM, 177 ± 6.7 kg), approximately 5 months of age, were randomly assigned to three rooms of eight pens and two rooms of four pens in an enclosed facility. After a 21 day adaptation, the calves were randomly assigned to treatment groups of eight calves per treatment. Calves were fed one of four dietary treatments in individual pens for at least 84 days either: (1) control corn + supplemental pellet (CC), (2) control corn + YCW supplemental pellet (CY), (3) contaminated corn + supplemental pellet (MC), and (4) contaminated corn + YCW supplemental pellet (MY) (Table 1). Calves were fed diets containing low or high levels of mycotoxin contamination with or without the inclusion of YCW (Alltech Inc., Nicholasville, KY, USA) at 10 g/kg (DM basis) of the total ration. Diets consisted of (DM basis) 750 g/kg whole corn grain and 250 g/kg supplemental pellet with corn as the source of food-borne mycotoxins. Diets were formulated to meet or exceed requirements as set by NRC (1996) and pellets were individually formulated for each

Table 1
Diet composition, analysis and mycotoxin concentrations.

Ingredient composition ^b	Dietary treatment ^a			
	CC	CY	MC	MY
Control corn	750	750	–	–
Mycotoxin contaminated corn	–	–	750	750
Supplemental pellet	250	250	250	250
<i>Nutrient composition^c</i>				
DM	891	891	901	899
CP	187	196	206	216
aNDF	115	105	107	106
ADF	38	39	41	38
<i>Mycotoxin contamination^{d,e}</i>				
DON	0.50	0.48	9.74	10.79
15-Acetyl DON	<0.02	<0.02	1.25	1.28
ZEN	<0.02	<0.02	1.86	1.81
T-2 toxin ^f	<0.02	<0.02	0.44	0.33
HT-2 toxin ^g	<0.02	<0.02	0.44	0.33

DON, deoxynivalenol; ZEN, zearalenone.

^a Dietary treatment: CC = control corn and control pellet ($n=7$); CY = control corn and YCW pellet ($n=8$); MC = mycotoxin contaminated corn and control pellet ($n=7$); MY = mycotoxin contaminated corn and YCW pellet ($n=8$).

^b g/kg (DM basis).

^c Average of pooled samples ($n=4$) in g/kg of diet DM.

^d Average of pooled samples ($n=4$) in mg/kg of diet DM.

^e Concentrations of 3-acetyl-DON, diacetoxyscirpenol, T-2 triol, iso T-2 toxin, scirpentriol, nivalenol, 15-acetoxyscirpentriol, neosolaniol, acetyl T-2 toxin, zearalenol, aflatoxin B1, fumonisin B1, T-2 teraol, and fusarenone-X in the pooled samples were below detection limits.

^f Average of pooled samples containing T-2 toxin; remaining pooled samples below detection limits.

^g Average of pooled samples containing HT-2 toxin; remaining pooled samples below detection limits.

Table 2
Supplemental pellet composition.

Pellet ingredient ^b	Dietary treatment ^a			
	CC	CY	MC	MY
Top soy ^c	540.8	520.0	892.0	876.0
Urea	40.0	40.0	20.4	18.0
Mineral premix ^d	1.6	2.0	2.0	2.0
Vitamin premix ^e	0.4	0.4	0.4	0.4
Monensin premix ^f	1.2	1.2	1.2	1.2
Limestone	80.0	63.6	78.0	55.2
Salt	6.0	6.0	6.0	7.2
YCW ^g	–	40.0	–	40.0
Control corn	330.0	326.8	–	–

^a Dietary treatment: CC = control corn and control pellet ($n = 7$); CY = control corn and YCW pellet ($n = 8$); MC = mycotoxin contaminated corn and control pellet ($n = 7$); MY = mycotoxin contaminated corn and YCW pellet ($n = 8$).

^b g/kg DM basis of pellet.

^c Rumen escape soybean meal (Shur-gain, Guelph, Ontario, Canada).

^d Contained 153,013 mg/kg of Zn, 122,445 mg/kg of Mn, 30,598 mg/kg of Cu, 27,100 mg/kg of Fe, 368 mg/kg of Co, and 1531 mg/kg of I.

^e Contained 4,400,000 IU/kg of vitamin A, 1,100,000 IU/kg of vitamin D, and 7700 IU/kg of vitamin E.

^f Contained 200 g of monensin/kg.

^g YCW is a modified yeast cell wall extract (Alltech Inc., Nicholasville, KY, USA).

treatment (Table 2) so that diets would be isonitrogenous (CP, 180 g/kg DM) and isoenergetic (diet metabolizable energy, 12.7 MJ/kg). Diets were provided to the calves for *ad libitum* intake and each calf had access to individual automated drinking fountains. The project was approved by the University of Guelph Animal Care Committee and followed recommendations of the Canadian Council on Animal Care (1993).

2.2. Measurement of body weight and feed consumption

Calves were weighed before feeding every 21 days on an electronic scale. Calves were weighed on two consecutive days at the beginning and end (*i.e.*, day 84) of the experiment and the weights were averaged to obtain initial and final BW during the test period. Feed amounts were measured at the time of feeding prior to being fed to the calves and individually recorded daily. Orts remaining in each feeder were weighed and recorded weekly. Feed samples were collected weekly and composited over 21 day periods. Composited samples were analyzed for nutrient content at Agri-Food Laboratories (Guelph, ON, Canada). DM analysis was in accordance with the Association of Official Analytical Chemists guidelines (AOAC, 1990, Method 930.15). Crude protein (CP) was determined by multiplying N by 6.25 as determined by Leco N analyzer (Leco Corporation, St. Joseph, MI, USA). aNDF (assayed with heat stable amylase and sodium sulphite, and expressed inclusive of residual ash) and ADF (expressed inclusive of residual ash) were determined using the methods of Robertson and Van Soest (1981) using an Ankom fibre analyzer (Ankom Technology Corp., Macedon, NY, USA).

2.3. Analysis of feedborne mycotoxins

Dietary contents of deoxynivalenol (DON), 3-acetyl-DON, 15-acetyl-DON, diacetoxyscirpenol, T-2 triol, T-2 toxin, iso T-2 toxin, scirpentriol, nivalenol, 15-acetoxyscirpentriol, neosolaniol, HT-2 toxin, acetyl T-2 toxin, zearalenol, zearalenone, aflatoxin B1, fumonisin B1, T-2 teraol and fusarenone-X were analyzed using a combination of gas chromatography–mass spectrometry (Raymond et al., 2003) and high performance liquid chromatography (Agilent 1100 Series, Agilent Technologies, Santa Clara, CA, USA) at the Veterinary Diagnostic Laboratory of North Dakota State University (Fargo, ND, USA). Composited feed samples (per 21 day period) were ground before analysis. The detection limit was 0.02 mg/kg for all toxins except for fumonisin B1 which had a detection limit of 2.0 mg/kg.

2.4. Blood collection, biochemistry and serum immunoglobulins

Blood samples were collected prior to feeding on days 0, 21, 42, 63 and 84 via jugular venipuncture. Plasma and serum were harvested by centrifugation ($2500 \times g$ for 15 min at 4 °C) and stored at –20 °C until analysis. Plasma concentrations of glucose (Trinder, 1969) and urea N (PUN; Sampson et al., 1980) were determined by spectrophotometry using a PowerWave XS microplate spectrophotometer (BioTek Instruments Inc., Winooski, VT, USA) and commercially available kits from Teco Diagnostics (Anaheim, CA, USA). Plasma concentrations of fibrinogen (Amelung KC4delta; Somagen Diagnostics, Edmonton, AB, Canada) and serum concentrations of haptoglobin (Hitachi 911 autoanalyzer; Roche Diagnostics, Division of Hoffman-La Roche Limited, Laval, QC, Canada) were determined by the Animal Health Laboratory of the University of Guelph. Serum concentrations of immunoglobulin A (IgA) and immunoglobulin G (IgG) were determined using commercially available ELISA kits (Bethyl Laboratories, Inc., Montgomery, TX, USA).

2.5. Slaughter and collection of organ weights

Calves were slaughtered after at least 84 days on treatment over a period of 8 days at the University of Guelph abattoir (Department of Animal and Poultry Science) and slaughter was ordered so that the heaviest animals from each treatment were killed first. Liver, kidney, heart and lungs were excised and weighed on an electronic scale. Organ weights were expressed as absolute organ weight (g) and relative to BW (g/kg).

2.6. Measurement of live animal and carcass characteristics

Ultrasound measurements of back fat depth (BF, mm), longissimus muscle area (LMA, cm²), and marbling score (1 devoid – 11 Prime+ according to Canadian beef quality grade) were performed (Montanholi et al., 2009) on the calves on days 0, 42 and 84 of the experiment using an Aloka SSD-500 ultrasound unit (Corometrics Medical Systems, Wallingford, CT, USA). At slaughter, hot carcass weights (HCW) were recorded without the kidneys and kidney fat. At 2 days post-mortem, calves were inspected by an experienced carcass evaluator from the Canadian Beef Grading Agency following the Livestock and Poultry Carcass Grading Regulations (Canada Gazette, 1992). The interface between the 11th and 12th ribs was used to obtain LMA (cm²), and mm of BF depth (minimum depth of back fat in the last quadrant over the longissimus muscle). The fresh cut surface of 2 day aged steaks were exposed to air to allow exposure to atmospheric oxygen for 30 min to enhance colour development before using a Konica Minolta Chroma Meter (Model CR-400; Mississauga, ON, Canada) to measure lightness or luminosity (*L**value); with a higher value indicative of a lighter colour.

2.7. Statistical analyses

The experiment was analyzed as a randomized complete block with a 2 × 2 factorial arrangement of treatments using the MIXED procedure of SAS (2008). The model included effects of the block (room), level of contamination, presence of the YCW and the interaction of the mycotoxin contaminated diets (M) and YCW diets (Y). Two animals died, one from CC (day 75; internal haemorrhage) and one from MC (day 78; pH related causes), and lack data points (*n* = 7) for the final collection periods. Measurements of ADG, feed:gain (F:G), DM intake (g FI/kg BW) and blood analytes were analyzed using repeated measures analysis within the MIXED procedure of SAS. Data from the two calves that did not finish were used in the repeated measures analysis up until their death. Covariance models for these measurements were chosen based on the lowest value for the Akaike information criteria fit statistics as outlined by Wang and Goonewardene (2004). Statistical significance was declared at *P* ≤ 0.05 for all models.

3. Results

3.1. Analysis of feedborne mycotoxins

The major contaminant in contaminated diets was deoxynivalenol with concentrations of 15-acetyl DON, zearalenone, T-2 toxin and HT-2 toxin also above detectable levels in both the MC and MY treatments (Table 1). The levels of DON, 15-acetyl DON, zearalenone, T-2 toxin and HT-2 did not differ between MC and MY treatments (Table 1). DON was also detected in the CC and CY treatments with average concentrations of 0.51 mg/kg DM and 0.48 mg/kg DM, respectively. The average concentration of DON in the MC treatment was 9.74 mg/kg DM and in the MY treatment was 10.79 mg/kg DM.

3.2. Performance

Final BW of the calves, as well as total BW gain and DM intake (g FI/kg BW) were not different between the treatment groups (Table 3). However, ADG tended (*P* = 0.07) to be higher in calves fed the high mycotoxin diets. There was an M*Y interaction (*P* = 0.04) for DM intake (g FI/kg BW) with DM intake decreasing with addition of YCW in the control treatments and increasing in the contaminated treatments. Feeding the high mycotoxin diets also resulted in decreased F:G (*P* = 0.003).

3.3. Blood analysis

Acute phase proteins, haptoglobin and fibrinogen, as well as concentrations of IgA did not differ between treatments (Table 4). Concentrations of IgG were lower (*P* = 0.02) in calves fed the YCW. PUN and plasma glucose concentrations were increased (*P* < 0.001) and decreased (*P* = 0.004), respectively, in calves fed the high mycotoxin diets.

3.4. Organ weights and carcass characteristics

Weights (g and g/kg BW) of the liver, kidney, heart and lungs did not differ between treatments (Table 5). Change in LMA, marbling score and BF, as indicated by ultrasound, from the initial to final measurements, were not affected by diet (Table 6). At slaughter, HCW, dressing yield, BF, LMA and colour Minolta *L**value were not different between treatments (Table 7).

Table 3

Effect of feeding corn naturally contaminated with *Fusarium* mycotoxins and modified yeast cell wall extract on veal calf performance, dry matter intake and feed efficiency.

	Dietary treatment ^a				SEM	P		
	CC	CY	MC	MY		M	Y	M*Y
Initial BW (kg)	176	181	173	177	6.7	0.58	0.51	0.92
Final BW (kg)	314	320	313	325	11.0	0.85	0.42	0.78
BW gain ^b (kg)	140	138	141	148	5.9	0.38	0.63	0.47
ADG (kg/day)	1.61	1.63	1.67	1.77	0.060	0.07	0.35	0.46
DM intake (g FI/kg BW)	26.6	25.7	25.6	26.3	0.41	0.51	0.77	0.04
F:G	4.32	4.10	3.86	3.80	0.140	0.003	0.27	0.54

ADG, average daily gain; BW, body weight; F:G, feed:gain; M, mycotoxin contaminated diets; Y, modified yeast cell wall extract (YCW) diets.

^a Dietary treatment: CC = control corn and control pellet ($n = 7$); CY = control corn and YCW pellet ($n = 8$); MC = mycotoxin contaminated corn and control pellet ($n = 7$); MY = mycotoxin contaminated corn and YCW pellet ($n = 8$). Values are least square means and SEM ($n = 7$) for M*Y interaction.

^b Total weight gain over the 84 days on treatment.

Table 4

Effect of feeding corn naturally contaminated with *Fusarium* mycotoxins and modified yeast cell wall extract on blood analytes.

	Dietary treatment ^a				SEM	P		
	CC	CY	MC	MY		M	Y	M*Y
Haptoglobin (mg/dL)	12.3	12.6	12.3	12.2	1.09	0.15	0.66	0.18
Fibrinogen (mg/dL)	366	351	356	334	17.6	0.41	0.26	0.85
IgA (mg/dL)	19.1	18.1	18.8	17.6	2.25	0.86	0.60	0.98
IgG (g/dL)	10.9	8.5	11.0	9.4	0.91	0.58	0.02	0.68
Plasma glucose (mg/dL)	98.6	98.5	94.4	95.1	1.45	0.004	0.79	0.75
PUN (mg/dL)	9.8	10.6	11.8	11.6	0.45	<0.001	0.47	0.24

IgA, immunoglobulin A; IgG, immunoglobulin G; M, mycotoxin contaminated diets; PUN, plasma urea N; Y, modified yeast cell wall extract (YCW) diets.

^a Dietary treatment: CC = control corn and control pellet ($n = 7$); CY = control corn and YCW pellet ($n = 8$); MC = mycotoxin contaminated corn and control pellet ($n = 7$); MY = mycotoxin contaminated corn and YCW pellet ($n = 8$). Values are least square means and SEM ($n = 7$) for M*Y interaction.

Table 5

Effect of feeding corn naturally contaminated with *Fusarium* mycotoxins and modified yeast cell wall extract on organ weights.

	Dietary treatment ^a				SEM	P		
	CC	CY	MC	MY		M	Y	M*Y
Liver								
kg	5.93	6.02	5.77	5.83	0.256	0.49	0.76	0.96
g/kg BW	18.1	18.2	17.6	17.4	0.61	0.25	0.92	0.83
Lung								
kg	4.69	4.16	4.35	4.85	0.348	0.61	0.96	0.14
g/kg BW	14.4	12.6	13.4	14.5	1.06	0.65	0.68	0.17
Kidney								
kg	1.19	1.14	1.15	1.13	0.055	0.65	0.52	0.77
g/kg BW	3.66	3.45	3.51	3.39	0.139	0.44	0.25	0.75
Heart								
kg	1.53	1.52	1.66	1.49	0.074	0.51	0.22	0.29
g/kg BW	4.71	4.61	5.04	4.46	0.237	0.70	0.14	0.30

BW, body weight; M, mycotoxin contaminated diets; Y, modified yeast cell wall extract (YCW) diets.

^a Dietary treatment: CC = control corn and control pellet ($n = 7$); CY = control corn and YCW pellet ($n = 8$); MC = mycotoxin contaminated corn and control pellet ($n = 7$); MY = mycotoxin contaminated corn and YCW pellet ($n = 8$). Values are least square means and SEM ($n = 7$) for M*Y interaction.

Table 6

Effect of feeding corn naturally contaminated with *Fusarium* mycotoxins and modified yeast cell wall extract on change in ultrasound carcass characteristics^a.

	Dietary treatment ^b				SEM	P		
	CC	CY	MC	MY		M	Y	M*Y
BF (mm)	0.72	0.74	0.72	0.53	0.180	0.54	0.61	0.55
LMA (cm ²)	14.3	14.8	14.7	16.5	1.81	0.55	0.49	0.71
Marbling score ^c	0.34	0.14	0.42	0.35	0.233	0.52	0.55	0.77

BF, back fat; LMA, longissimus muscle area; M, mycotoxin contaminated diets; Y, modified yeast cell wall extract (YCW) diets.

^a Change is the difference between the final value (day 84) and the initial value (day 0).

^b Dietary treatment: CC = control corn and control pellet ($n = 7$); CY = control corn and YCW pellet ($n = 8$); MC = contaminated corn and control pellet ($n = 7$); MY = contaminated corn and YCW pellet ($n = 8$). Values are least square means and SEM ($n = 7$) for M*Y interaction.

^c 1:devoid – 11: Prime+ according to Canadian beef quality grade.

Table 7Effect of feeding corn naturally contaminated with *Fusarium* mycotoxins and modified yeast cell wall extract on post-mortem carcass characteristics.

	Dietary treatment ^a				SEM	P		
	CC	CY	MC	MY		M	Y	M*Y
HCW (kg)	177	176	171	177	4.7	0.64	0.52	0.46
Dressing yield ^b	563	553	546	547	8.6	0.18	0.56	0.52
BF (mm)	2.81	2.87	2.11	3.62	0.762	0.97	0.29	0.33
LMA (cm ²)	48.3	49.6	47.2	48.1	1.69	0.44	0.51	0.92
Colour (L*value)	38.9	38.2	38.6	37.6	0.61	0.43	0.15	0.78

BF, back fat; LMA, longissimus muscle area; HCW, hot carcass weight; M, mycotoxin contaminated diets; Y, modified yeast cell wall extract (YCW) diets.

^a Dietary treatment: CC = control corn and control pellet ($n=7$); CY = control corn and YCW pellet ($n=8$); MC = mycotoxin contaminated corn and control pellet ($n=7$); MY = mycotoxin contaminated corn and YCW pellet ($n=8$). Values are means and SEM ($n=7$) for M*Y interaction.^b g carcass weight/kg BW.

4. Discussion

4.1. Performance

The feeding of diets naturally contaminated with *Fusarium* mycotoxins often results in feed refusal or decreased feed intake in sensitive species such as swine and poultry (Swamy et al., 2003; Chowdhury and Smith, 2004). Throughout the experiment, the DM intake of the calves generally was not affected by the presence of DON, 15-acetyl DON and ZEN in the feed. This corresponds to results of Korosteleva et al. (2007, 2009) in dairy cows fed a naturally contaminated diet where no feed intake differences occurred. The decreased feed intake observed in other species contributes to decreased BW gains in animals fed contaminated diets. In our study, there was no difference in total BW gain among treatments, although the calves fed contaminated corn had a tendency ($P=0.07$) to a higher ADG than calves fed the control corn. This could potentially be due to the presence of the estrogenic mycotoxin zearalenone, which may influence growth. In the rumen, zearalenone is transformed into α -zearalenol and β -zearalenol, both of which are more estrogenically active than the parent toxin (Dänicke et al., 2005). Also, although all diets were formulated to be isonitrogenous, the calves fed contaminated corn received numerically more CP than calves fed the control corn. However, statistical analysis of dietary composition of composited samples indicated that the CP concentration did not differ among treatments (Table 1) and all diets were above CP requirements (NRC, 1996), making it unlikely that differences in CP intake influenced performance. Dänicke et al. (2002) also noted higher CP levels in the contaminated ration fed to growing bulls (average BW 352 kg), which corresponded to higher BW gains at the beginning of their study. In our experiment, the tendency to higher ADG, with no difference in DM intake contributed to the improved F:G in calves fed contaminated diets. Swamy et al. (2003) also observed an improvement in feed efficiency in swine fed a contaminated diet. Conversely, in that experiment, concurrent reductions in feed intake may have caused better utilization of nutrients resulting in the improved efficiency of growth and feed utilization.

4.2. Blood analysis

The immune effects of *Fusarium* mycotoxins can vary depending on the level of mycotoxin in the feed and the sensitivity of the species fed the contaminated feedstuff. Low levels of these mycotoxins can stimulate the immune system, typically through gastrointestinal tract inflammation (Pestka, 2008). Acute phase proteins are useful indicators of trauma, infection and inflammatory processes (Petersen et al., 2004). Concentrations of both of the acute phase proteins haptoglobin and fibrinogen were not affected by the presence of DON, 15-acetyl DON and ZEN, suggesting that the concentrations in the diets did not cause an inflammatory response through an activation of acute phase proteins. Korosteleva et al. (2007) also reported no response in the acute phase protein haptoglobin with feeding of mycotoxin contaminated grains to dairy cows.

Consumption of *Fusarium* mycotoxins can result in suppression of the immune system, through apoptosis of immune cells and organs, up-regulation and down-regulation of important genes involved in the immune system, alteration of oxidative pathways and/or disruption of physical barriers (Pestka, 2008). Lack of an effect of the high mycotoxin diets on concentrations of immunoglobulins suggests DON, 15-acetyl DON and ZEN did not alter the immune system in regards to the measured immunoglobulins. However, our calves were likely not exposed to significant levels of stress or disease, which could be more likely to occur in commercial conditions, and results could differ depending on management and environmental conditions. However, use of an YCW in our study resulted in lower serum concentrations of IgG. Research on YCW suggests that it can have a mixed response on alleviating the immune effects of mycotoxins, either preventing effects on the immune system (Chowdhury et al., 2005b) or having no effect (Chowdhury et al., 2005a; Korosteleva et al., 2007).

The higher concentrations of PUN noted calves fed the contaminated grain source may, in part, be due to the higher levels of CP observed with the MC and MY diets (Table 1). However, a study with dairy cattle suggested that animals receiving *Fusarium* mycotoxins had elevated concentrations of serum urea in circulation (Korosteleva et al., 2007). They hypothesized that rumen microbial protein synthesis may be inhibited in rumen microbes and protein synthesis inhibited in animal tissues in animals exposed to these mycotoxins, which could result in higher levels of free ammonia and amino acids in blood supplying the liver, respectively, and consequently increase urea N concentration in the blood.

Blood glucose is not as tightly regulated in young ruminants as compared to older ruminants and can fluctuate widely in the first year of age (Kennedy et al., 1939). This may have made the calves more sensitive to small dietary differences in carbohydrates thereby contributing to the lower levels of plasma glucose in the MC and MY diets. The presence of fungi on crops can cause the plant to alter its chemical and physical properties which may change the structure of the carbohydrate component of the diet (Brinkmeyer et al., 2006). In turn this could alter ruminal volatile fatty acid production, digestibility and nutrient absorption, which also could influence plasma glucose concentrations.

4.3. Carcass characteristics

The carcass weights, muscling and fat cover were not affected by treatment. As expected with calves of this size, the marbling and BF deposition were very small, making differences difficult to detect. An important aspect of veal quality that impacts the final grade is meat colour with a lighter, pink coloured lean tissue desired with darker, red colour lean tissue receiving lower grades. Colour, as measured by the industry standard Minolta colour reflectance meter, was also not influenced by treatment. However, research in poultry suggests that meat colour may be adversely affected by the presence of mycotoxins in the diet (Wang et al., 2006). Overall, the carcass characteristics of the calves were not impacted by the presence of DON, 15-acetyl DON and ZEN or YCW in the diet.

5. Conclusion

Veal calves are able to tolerate a moderate feeding level of corn grain naturally contaminated with *Fusarium* mycotoxins (DON, 15-acetyl DON and ZEN) and small improvements in F:G were observed. However, as the reaction of calves greatly depends on the compounds, concentration, duration of exposure and combinations of different mycotoxins in the diet, the feeding of contaminated corn grain to veal calves needs to be done with caution. As there were generally no negative effects of dietary mycotoxin level, the efficacy of YCW as a mycotoxin adsorbent could not be assessed, but YCW alone had negligible effects on performance.

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