

2019 TVMDL Amarillo



# CATTLE HEALTH MANAGEMENT CONFERENCE

## FUMONISIN EXPOSURE AND CATTLE PERFORMANCE IN THE TEXAS HIGH PLAINS

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### INTRODUCTION

Fumonisin is a mycotoxin produced by molds of the genus *Fusarium*. While these toxins can be found in many agricultural crops, corn is the major commodity affected (FDA, 2001a). The specific causes of increased fumonisin levels in corn are still unknown, but may include drought stress followed by warm, wet weather later in the season (Richards, 2007), though insect damage and improper post-harvest storage are also potential contributors (FDA, 2001b). Fumonisin exposure has the potential for negative impacts on human and livestock health, such as leukoencephalomalacia in horses, lung edema in swine, and it may also contribute to certain human cancers (Richards, 2007).

To address and mitigate the risk posed by fumonisin in human and livestock feeds, in 2001 the United States Food and Drug Administration released a guidance document identifying maximum fumonisin levels for corn and corn byproducts considered adequate to protect human and livestock health (FDA, 2001b). These guidance levels are considered achievable with use of good agricultural and manufacturing practices and are based on the results of prior research into health and performance impacts of fumonisin. These guidelines take into account species-specific susceptibilities to fumonisin, with humans and rabbits deemed the most sensitive and cattle, mink, and poultry the least sensitive (FDA, 2001b).

For ruminants, available data on the effects of fumonisin exposure are relatively limited (FDA, 2001b). In particular, guidelines for ruminants are influenced by the study of Osweiler et al. (1993), who fed diets containing < 5, 24.6, and 110.3 ppm of dietary fumonisin to feeder calves for 31 days, followed by 30 days on a control diet. While Osweiler et al. (1993) found reversible liver and immunological impacts of fumonisin exposure in feeder calves on the diet highest in fumonisin, there were no significant impacts on performance and the authors recommended that further research be conducted into any possible negative impacts of feeding cattle higher doses of fumonisin for longer periods of time.

Based on these results, as well as supporting evidence from other studies, the FDA concluded that 120 ppm dietary fumonisin was likely hazardous to most ruminants (FDA, 2001b). However, gaps in the scientific literature regarding exposure of ruminants to between 25 and 120 ppm total dietary fumonisin led to establishment by FDA of a conservative limit of 60 ppm fumonisin in corn for feeding to ruminants. Any corn containing 60 ppm fumonisin should be limited to ≤ 50% of the total diet on a dry matter basis, for a final diet fumonisin content of ≤ 30 ppm for ruminants older than 3 months old. To prevent carcinogenic effects in breeding stock, this guidance level was set lower, at 15 ppm dietary fumonisin, based primarily on chronic exposure data from mice and rats (FDA, 2001b).

To address these gaps in current scientific knowledge, the objective of the present study was to investigate the impact of fumonisin exposure on feedlot cattle intake and performance, with the hypothesis being that dry matter intake would decrease as dietary fumonisin levels and length of exposure increased.



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## MATERIALS AND METHODS

Fifty steers were received (d 0), vaccinated, implanted (Revalor-200, 200 mg trenbolone acetate and 20 mg estradiol, Merck Animal Health) and dewormed (Dectomax pour-on, Zoetis). One steer was removed from the study due to a foot injury, leading to a final number of 49 steers on trial. Animals were housed individually in pens and fed once daily at 0800 h, and bunks were divided to prevent cross-contamination between treatment diets fed in adjacent pens. Steers were transitioned to a finishing diet (Table 1) from d 0 to 21 and then were fed the control finishing diet ( $\leq 5$  ppm fumonisin) until d 50, when initiation of feeding treatment diets containing graded levels of fumonisin began. Steers were fed the fumonisin treatment diets from d 50 until slaughter at d 160, and individual animal body weights were taken on days 0, 50, 100, 150 and 160. Steers were re-implanted on d 100 (Revalor-200, 200 mg trenbolone acetate and 20 mg estradiol, Merck Animal Health)

Cattle were fed a dry-rolled corn-based finishing diet (Table 1). Treatment diets were formulated to achieve  $\leq 5$  ppm (**CON**), 15 (**15PPM**), 30 (**30PPM**), 60 (**60PPM**), and 90 ppm (**90PPM**) of total dietary fumonisin. Two corn supplies, one with little to no fumonisin (**NOFUM**) and one with high levels of fumonisin (**FUM**), were identified for the research. The corn supplies were rolled, transported to the feedlot, and stored in separate agriculture bags. During unloading of the grain at the feedlot, grain samples were collected from each truck as the dry-rolled corn was placed into the agriculture bag. The average of each sample was used to calculate the appropriate fumonisin level needed in the corn mixtures and treatment diets. Each corn mixture was batched separately by mixing NOFUM with FUM to achieve the desired fumonisin level. Treatment diets were batched daily using the appropriate corn mixtures. Daily samples of each diet were collected and composited weekly by treatment. The average fumonisin concentration of weekly composited samples was used to calculate final fumonisin exposure levels.

When approximately 60% of cattle on trial were expected to grade USDA choice, they were transported to Tyson Fresh Meats (Amarillo, TX) for harvest. This occurred at d 160 (110 d on treatment diets). Individual hot carcass weight was recorded immediately, while fat thickness, KPH fat, marbling score, ribeye, quality and yield grade were determined after a 48-h chill. Livers were examined for presence of abscesses, telangiectasia, and any other defects.

Animal performance and carcass data were analyzed using PROC MIXED in SAS (SAS Institute, Cary, NC) with animal as the experimental unit, and PROC GLIMMIX was used to analyze categorical carcass data. Contrasts were used to test for linear and quadratic responses. Quadratic responses were not detected therefore only linear responses were reported. Effects were considered significant at  $P \leq 0.05$  and tendencies were declared at  $0.05 < P \leq 0.10$ .

## RESULTS & DISCUSSION

An indirect objective of this study was to determine whether spot testing of individual loads of grain provided an accurate estimate of the amount of fumonisin in a larger supply of grain. The actual dietary fumonisin levels achieved are presented in Table 2. The CON treatment averaged 8.1 ppm dietary fumonisin, while 15PPM, 30PPM, 60PPM, and 90PPM treatments averaged 26.7, 40.7, 67.6, and 108.8 ppm dietary fumonisin, respectively. Therefore, spot sampling a larger corn supply does represent the fumonisin levels throughout the supply.



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Throughout the study, there was no effect of treatment ( $P > 0.60$ ), or a linear response ( $P > 0.16$ ), on BW or DMI (Table 3). However, CON tended to have lower ADG than other treatments during the treatment period ( $P = 0.10$ ), and there was a linear response ( $P = 0.02$ ) of ADG to fumonisin dose during the treatment period. Similarly, ADG tended to respond linearly over the entire feeding period ( $P = 0.06$ ), as did F:G ( $P = 0.07$ ). These results indicate that increasing levels of dietary fumonisin does not adversely affect feedlot cattle performance.

The results of this study are in accordance with prior results from Osweiler et al. (1993), who reported no differences in BW, ADG, or DMI among calves fed three treatments of  $< 5$  ppm, 24.6 ppm, and 110.3 ppm dietary fumonisin for 31 days, followed by 30 days on the control diet ( $< 5$  ppm dietary fumonisin). Osweiler et al. (1993) further suggested that while it appeared that cattle were relatively less susceptible to fumonisin exposure than swine or horses, the effects of fumonisin for longer feeding periods remained to be determined. The results of this study, in which cattle were fed fumonisin for 110 days, suggests that the reduced susceptibility of cattle vs. swine and horses is true even during longer feeding periods as well.

There were no differences between treatments with regard to HCW, DP, marbling score, ribeye area, or calculated YG (Table 4). However, a linear effect occurred for both fat thickness ( $P = 0.04$ ) and percent KPH ( $P = 0.03$ ), with both decreasing as dietary fumonisin dose increased. The exact cause of this decrease in fat deposition remains unknown.

During slaughter, livers were scored for the presence of abscesses as well as for telangiectasis and any other anomalies. Telangiectasis is a liver lesion characterized by the appearance of depressed, reddish-brown spots on the liver surface (Atasever et al., 2002; Yousef et al., 2011). While several potential causes have been identified such as, inflammation of the hepatic portal system and liver, exposure to toxins, and inadequate blood supply to liver cells, the underlying cause is still unknown (Jensen et al., 1982, Yousef et al., 2011). There were no effects of treatment on either liver abscesses ( $P = 0.95$ ) or telangiectasis ( $P = 0.13$ ; Table 4). It has been hypothesized that telangiectasis and liver abscesses were correlated, and perhaps that telangiectasis predisposed cattle to later abscesses; however, Atasever et al. (2002) found no correlation between the two conditions.

Osweiler et al. (1993) examined blood indicators of liver function in cattle exposed to fumonisin. The authors noted that while changes were observed in cattle fed the high fumonisin diet relative to the control animals, these changes were not enough to indicate severe liver disease. Observed changes to liver function and physiology, as well as spleen physiology, were also considered mild and reversible once exposure to fumonisin ended, as by 30 days on the control diet ( $< 5$  ppm dietary fumonisin) liver chemistry was not different among treatments (Osweiler et al., 1993). Consistent with the blood chemistry results, changes to liver physiology in animals fed fumonisin were also observed upon slaughter indicating mild to moderate elevation of liver enzymes (Osweiler et al., 1993). The impacts of fumonisin exposure on liver function and appearance observed in Osweiler et al. (1993) may not be significant for heavier cattle fed longer periods of time. We speculate that any initial effects on liver function may be reversible over time, perhaps as animals develop further resistance to the effects of fumonisin.



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## CONCLUSION

No differences in performance, carcass, or liver characteristics were observed in steers fed dietary levels of fumonisin ranging from 8 to 108 ppm for 110 days prior to harvest. The results of this study agree with previous results that reported no differences in performance among calves fed increasing dietary fumonisin levels for 31 days. The results of this study support the theory of reduced susceptibility of cattle to the effects of fumonisin and suggests that this reduced susceptibility may hold true for cattle fed to heavier final weights and for longer feeding periods, such as in a commercial feedlot setting.

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