No brainers: Understanding and mitigating sulfur toxicity in feedlot cattle

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Introduction

Increased intake of S has been shown to decrease intake, reduce gain and can lead to a neurological disorder called polioencepahlamalacia (PEM), commonly referred to as polio or brainers. Sulfur, when fed to ruminants is reduced to toxic hydrogen sulfide (H_2S) by ruminal bacteria. The ruminal accumulation, eructation and inhalation of large amounts of H_2S is thought to be the cause of these negative effects. It is important to consider dietary S contributions from both water and feed ingredients when determining S intake by cattle. The maximum tolerable limit for S in beef cattle diets has been suggested to be 0.3% S in diets containing greater than 85% concentrate (NRC, 2005).

Sulfate reducing bacteria

Dissimilatory sulfate reducing bacteria (SRB) reduce sulfate and produce H_2S as an end product of their metabolism (Bradley et al., 2011). Many SRB can also reduce other oxidized inorganic sulfur compounds (sulfite, thiosulfate, or elemental sulfur) as well as S-containing amino acids (Colman, 1960). When genomic technology was utilized to determine the major SRB present when steers were fed 0.3% or 0.6% S finishing diets the top three genera of SRB were *Desulfovibrio, Desulfohalobium,* and *Sulfolobus* and were the same for both concentrations of S. However, the number of Desulfovibrio were 1.65 fold greater in the steers fed the 0.6% S diet compared to 0.3% S diet, while Desulfohalobium and Sulfolobus were relatively similar between dietary S treatments. The quantity of *Desulfovibrio desulfuricans* was found to be highly correlated with rumen H_2S concentrations and accounted for 60% of the variation in H_2S among steers as measured on d 155 (S. L. Hansen and E. L. Richter, unpublished). This supports an important role of *Desulfovibrio* in S metabolism in the ruminant and could be potential targets of mitigation strategies.

Ruminal hydrogen sulfide and polioencephalomalacia

Polioencephalomalacia is a neurologic disease of ruminants that results in lesions of the gray matter in the brain that was first associated with thiamine deficiency but has been found to be caused by other toxic or metabolic diseases such as high dietary S. Sulfur induced polioencephalomalacia (S-PEM) has been associated with elevated ruminal H_2S in experimental settings (Gould et al., 1997). Furthermore, Loneragan et al. (2005) found that S-PEM and the ruminal concentration of H_2S peaked at the same time in feedlot cattle consuming high sulfate water. It has been speculated that eructated H_2S gas that is inhaled by the animal can travel to the brain and then cause brain cell death. Dougherty and Cook (1962) observed that 70 to 80% of gas that is eructated from the rumen is subsequently inhaled by the ruminant, which would allow the eructated gases to enter circulation without being detoxified by the liver. Further, when Dougherty et al. (1965) infused H_2S into the rumen of sheep they observed that sheep with an open trachea collapsed after several eructations, whereas those with a blocked trachea produced no clinical signs suggesting that the mechanism of toxicity is not through ruminal absorption of H_2S, but through the inhalation of eructated H_2S.

Adaptation to finishing diets

Studies have suggested that H_2S concentrations increase during adaptation to a finishing diet containing increased concentrations of dietary S, with peak in H_2S production occuring somewhere between d 14 and 60 of the finishing period. Loneragan et al. (2005) utilized three concentrations of sulfate in the drinking water (136, 583, 2,360 mg sulfate/L) of feedlot cattle and observed that ruminal H_2S concentrations peaked on d 31 of the study, after which concentrations decreased and stabilized for the remainder of the trial. Drewnoski et al. (2012) found that cattle consuming a high S diet had concentrations of H_2S that were excessively increased relative to their S intake during the first 30 d on a finishing diet, regardless of whether or not cattle were previously exposed to elevated dietary S while on a high roughage diet. When steers were fed a high S finishing diet peak H_2S concentrations were observed from d 7 through 28 of the finishing period and this peak did not appear to be related to ruminal pH, as ruminal pH did not differ across the finishing period (Drewnoski et al., 2013). In their recent longitudinal analysis, Nichols et al. (2013) reported that approximately half of the PEM cases observed in their meta-analysis of finishing studies occurred during the first 60 d on feed (this included the step-up period). These data suggests that cattle adapting to finishing diets are at a greater risk for increased concentration of H_2S and the development of S-PEM.

Mitigation of toxicity

Currently, there is no magic bullet s in the battle against S toxicity. To date, the most valuable tools for nutritionists in the prevention of S-induced toxicity are an understanding of the ruminal availability of the S in the diet and inclusion of at least 7 to 8 % NDF from roughage. Recently, Sarturi et al. (2013) proposed the ruminally available S (**RAS**) as a method of better evaluating the potential of diets to result in production of ruminal H_2S. They suggested that S must be ruminally soluble in order to potentially contribute to H_2S production in the rumen and that organic sources of S such as S-amino would be less available for ruminal reduction by SRB than inorganic sources because some proportion of the dietary protein will be ruminally undegradable. Alternately, the excess S in co-products which comes from the use of sulfuric acid during processing would be completely available to SRB for reduction to sulfide. Similarly, other sources of inorganic S such as high sulfate water, calcium sulfate and ammonium sulfate would also be expected to be 100% available for reduction by SRB. When the RAS theory was examined, Sarturi et al. (2013) reported that RAS intake ($g \cdot steer^{-1} \cdot d^{-1}$) accounted for more of the variation in ruminal H_2S concentrations (65%) than total S intake (29%). Nichols et al. (2013) conducted a meta-analysis of finishing studies and suggested that there is a strong effect of the concentration of roughage NDF within a concentration of dietary RAS on the incidence of S-PEM. Risk of S-PEM decreased approximately 19% for each 1% increase in roughage NDF in the diet, within a given concentration of RAS.

While not as obvious as S-PEM, subacute S toxicosis in the form of decreased DMI and gains are economically detrimental. When effects on DMI and gain are evaluated cattle fed high forage diets appear to be more tolerant of increased concentrations of dietary S than cattle consuming high concentrate diets. Spears et al. (2011) observed that DMI was not affected in steers consuming a corn-silage based diet containing 0.12, 0.30, or 0.46% S but was linearly decreased when they switched to a ground corn based-diet. When the effect of S is studied within finishing diets containing no ethanol co-products (Zinn et al., 1997; Spears et al., 2011) or moderate amounts (30 or 40% on DM basis) of co-product (Uwituze et al., 2011a; Richter et al., 2012; Pogge and Hansen, 2013) a clear detrimental effect on DMI and gain is observed. Based on the weighted average slope of the effects observed in these studies the data suggests that for

every 0.1% increase in dietary S concentration beyond 0.2% S, a predicted decrease of 0.5 kg DMI and 0.126 kg ADG would occur.

Conclusions

If possible feedlot nutritionist's should wait to include high levels feedstuffs with elevated concentrations of S until after cattle have received their finishing diet for at least 28 days. With good bunk management and inclusion of 12 to 17% roughage (7 to 8% NDF from roughage) in the finishing diet feedlot nutritionists could be able to feed diets with 0.5% S (0.36% RAS) after the first 30 d of finishing.

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