The Professional Animal Scientist 31 (2015):101–108; http://dx.doi.org/10.15232/pas.2014-01364

REVIEW

REVIEW: Control of liver abscesses in feedlot cattle: A review[1]

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ABSTRACT

Published literature was reviewed to provide an overview of the historical prevalence and methods of controlling liver abscesses (LA) in feedlot cattle. Liver abscesses are typically categorized as mild, moderate, or severe, with severe LA most often being associated with reductions in performance. The prevalence of LA in beef-breed steers increased by 25% between 2008 and 2013; however, the prevalence in Holstein steers tripled over the same period. Regionally, the greatest prevalence has been observed in Kansas, eastern Colorado, and western Nebraska, and the lowest prevalence has been observed in the Midwest and the desert southwest. Fusobacterium necrophorum and Trueperella pyogenes are most commonly associated with LA, although F. necrophorum is likely the primary causative pathogen. Liver abscesses are often, but not always, associated with perforations in the rumen wall. Tylosin phosphate is commonly fed to control LA. Feeding elevated levels of roughage during growing and finishing periods results in a dramatic reduction in LA; overprocessing of dietary roughage reduces its effectiveness. Grain processing has marked effects on ruminal starch availability but has minimal effect on LA; inclusion of fibrous by-product feeds also does not mitigate prevalence of LA. Vaccination against F. necrophorum has shown little benefit in field application. Providing a source of true scratch-factor to the rumen, either by increasing the percentage of coarse roughage included in the TMR or by periodically providing coarse hay apart from the TMR, appears to be the most effective method of reducing LA.

Key words: cattle, feedlot, grain processing, liver abscess, roughage

INTRODUCTION

Liver abscesses (**LA**) are the primary cause of liver condemnation in feedlot cattle slaughtered in the United States, averaging 67% of all liver abnormalities (Brown and Lawrence, 2010). Feeding high-energy, grain-based finishing diets low in roughage, common in the feedlot industries in the United States, Canada, Mexico, Europe, South Africa, and Japan, is associated with elevated prevalence of liver abscesses (Nagaraja et al., 1996). Although all LA may affect animal performance to some degree, the most severe LA have the greatest effect and may reduce the value of beef carcasses by $38 per animal (Brown and Lawrence, 2010); in addition, there is concern for animal well-being in animals with severe liver abscesses. Because of the negative economic effect of LA, there is interest in prevention and control of LA in feedlot cattle. Nagaraja and Chengappa (1998) provided an excellent, thorough review of this topic. The objectives of the present review are to provide an update to that summary and to emphasize practical management factors germane to this issue.

PREVALENCE

Livers that are free from abscesses are classified as normal; livers categorized as having mild to moderate LA display ≤4 abscesses or resolved abscess scars ≤4 cm in diameter; livers categorized as having severe LA are livers displaying ≥1 abscesses >4 cm in diameter or >4 abscesses >2 cm in diameter (Elanco, 2014).

Prevalence of total LA (mild, moderate, and severe) ranges from close to 0% to greater than 70%, depending on several factors. However, the mean prevalence of total LA in conventionally managed United States feedlot cattle more commonly ranges from 10 to 20%, and the prevalence of severe LA commonly ranges from 4 to 6% (Davis et al., 2007; Brown and Lawrence, 2010; Rezac et al., 2014). Prevalence of LA varies by geographical location within the United States, with cattle slaughtered in the semi-arid plains of the central United States having 22 and 14% total and severe LA, respectively, and cattle slaughtered in the midwestern states, the southern plains, and the desert southwest averaging 13 and 4% total and severe LA, respectively, with the

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Pacific Northwest and the northern plains being intermediate averaging 19 and 7% total and severe LA, respectively (Elanco, 2014).

There has been a recent slight upward trend in the prevalence of LA in beef-breed steers, with an annual average that previously oscillated around 12% total LA from 2003 through 2008 rising steadily each year thereafter to 16% in 2013 (Elanco, 2014). However, the increase in prevalence of LA in Holstein steers has been much more noteworthy, rising steadily from 12% in 2003 and 2004 to 55% in 2013. Most of this increase has been observed in Holstein steers fed and slaughtered in the central high plains, where prevalence of LA in Holstein steers averaged 6 and 48% total and severe LA, respectively. Prevalence of LA in Holsteins fed and slaughtered in other parts of the United States has not changed dramatically, averaging 23 and 9% total and severe LA, respectively (Elanco, 2014). Prevalence also fluctuates seasonally, increasing roughly 4 percentage units for cattle slaughtered during the summer months versus those cattle slaughtered during the winter months (Elanco, 2014). Conversely, Brown and Lawrence (2010) reported that cattle slaughtered in the spring (March, April, and May) had a numerically greater percentage of liver condemnations for all causes and cattle slaughtered in the months of July, August, and September had numerically fewer liver condemnations.

One theory as to why calf-fed Holsteins suffer so much greater prevalence of LA versus beef-breed cattle is the extended duration of total days on feed. Calf-fed Holsteins, which typically arrive at feedlots weighing 130 to 180 kg and are fed a low-roughage, high-energy finishing diet for 300 to 400 d, have a much greater opportunity to develop LA versus their beef-breed counterparts, which typically arrive weighing 230 to 400 kg and are finished for only 120 to 240 d. Another theory has been proposed that management practices specific to the central plains region may contribute to increased prevalence of LA. This theory is unlikely because animal management has not changed significantly in the time period in question and because animal management does not differ substantially in the central, southern, and northern high plains. It has been suggested that LA organisms in calves raised in western calf ranches and subsequently shipped to feedlots in the central plains have developed resistance to tylosin. This theory is unlikely given the fact that LA prevalence has increased rapidly, has increased in non-Holstein, non-calf-ranch beef-breed cattle, and has not increased in other geographies where calves originating in these same calf ranches are also finished. Given the aforementioned evidence of a significant increase in LA in both beef breeds and in Holsteins, and that the increase has been observed only in the central high plains, over a short span of time, it is arguable that feedyards in the central high plains may have an infective pathogenic organism causing LA that simply is not extant in other geographic regions. This theory will require deeper investigation to substantiate or refute.

EFFECTS ON PERFORMANCE

Although mild and moderate LA have no or limited effects on animal performance (Davis et al., 2007; Fox et al., 2009; Brown and Lawrence, 2010), severe LA may reduce ADG by 0.06 to 0.20 kg (Brink et al., 1990; Fox et al., 2009; Rezac et al., 2014). Feed intake and feed conversion are also impeded by severe LA, reducing intake by 5% and gain-to-feed by 14% (Brink et al., 1990).

Presence of severe LA also may reduce HCW by 4 to 36 kg (Montgomery, 1985; Fox et al., 2009; Rezac et al., 2014). Severe LA, if associated with adhesion of the LA to the internal body-cavity wall results in even greater reduction in HCW. Davis et al. (2007) reported that the presence of severe LA was associated with 3.2-kg-lighter HCW but severe LA observed in conjunction with carcass adhesion was associated with 13.2-kg-lighter HCW; Brown and Lawrence (2010) reported that adhesions increased the loss in HCW by 3 kg in one comparison and by 8.7 kg in a second comparison. At least a portion of the increased loss in HCW may be trim; Montgomery (1985) reported an increase in trim loss of 0.43 percentage units for carcasses with severe LA, and Brown and Lawrence (2010) reported that carcasses with severe LA had 0.26 percentage units lower DP, possibly attributable to trim loss.

Carcass quality may also be reduced in cattle with severe LA. Whereas Davis et al. (2007) reported no difference in marbling score between cattle with normal livers and those with LA, Fox et al. (2009) reported that 7 percentage units fewer cattle graded Choice if severe LA were present versus cattle with normal livers. Brown and Lawrence (2010) reported a reduction in marbling score but also reductions in YG, fat depth, and percent KPH fat in cattle with severe LA versus cattle with normal livers.

Based on a reduction in carcass weight of 10 kg for cattle with severe LA versus cattle with normal, healthy livers, and the current carcass value of beef in the United States of $5.21/kg carcass weight (USDA, 2014), each animal with severe LA has reduced value of more than $52. If potential reductions in feed efficiency and carcass quality are also considered, the total loss in value is potentially even greater.

ETIOLOGY AND PATHOLOGY

Several different bacteria are commonly isolated from LA, including *Fusobacterium necrophorum* (Berg and Scanlan, 1982), *Trueperella pyogenes* (Calkins and Scrivner, 1967), *Bacteroides* spp. (Berg and Scanlan, 1982), *Clostridium* spp. (Simon and Stovell, 1971), *Pasteurella* spp. (Simon and Stovell, 1971), *Peptostreptococcus* spp. (Berg and Scanlan, 1982), *Staphylococcus* spp. (Berg and Scanlan, 1982), and *Streptococcus* spp. (Simon and Stovell, 1971), as well as other, not-yet-identified bacterial species, both gram-positive and gram-negative