THESIS

# LIVER ABSCESS EFFECTS ON CARCASS PERFORMANCE AND HERITABILITY ESTIMATES OF LIVER ABSCESS INCIDENCE AND SEVERITY IN BEEF ON DAIRY HEIFERS

Submitted by

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ABSTRACT

LIVER ABSCESS EFFECTS ON CARCASS PERFORMANCE AND HERITABILITY ESTIMATES OF LIVER ABSCESS INCIDENCE AND SEVERITY IN BEEF ON DAIRY HEIFERS

The economic impact of liver abscesses has been reported to be not only due to loss from condemnation of livers but also from impacts on performance. A primary focus in decreasing liver abscess prevalence has been on prevention methods because with limited or no clinical signs present, diagnosis of liver abscesses in live animals is complicated, and no prevention methods have been highly effective in mitigation. As a result, this study aimed to identify the impacts of liver abscesses on carcass performance and estimate heritability for liver abscess incidence and severity in fed beef on dairy heifers.

In the first study, 1,860 beef on dairy heifers were fed and harvested in Kansas. All had phenotypes for hot carcass weight (HCW; kg), rib eye area (REA; $cm^2$), fat thickness (FT; cm), marbling score (MS), calculated visual yield grade (VYG), and liver abscess score. Of the 1,860 individuals, 1,646 had phenotypes for heart score (HS). Carcass impacts were estimated using fixed effects of liver abscess score, contemporary group, and age in days. The contemporary group was a concatenation of kill lot and treatment. Liver abscess score was fit in two different forms: 6 scores (“0”, “A-”, “A”, “A+”, “A+AD”, “A+O”) and 4 scores (“0”, “A-”, “A”, “A+”) where “A+” included scores of “A+AD” and “A+O”. A score of “0” indicated no abscess and abscess severity increases with “A-”, “A”, and “A+”. The scores of “A+AD”, and “A+O” indicate there is adhesion of the liver to nearby organs and ruptured abscess, respectively. A

significant increase was identified using the six-score model for FT for animals with scores of "A+O" compared to "A+", with respective least-squares means of 1.94 cm ± 0.12 and 1.59 cm ± 0.06 ($P < 0.05$). While not significant, tendencies were identified for FT for animals with scores of "A" and "A+AD" compared to "A+O" ($0.05 \leq P < 0.1$) with respective least-squares means of 1.61 cm ± 0.06, 1.61 cm ± 0.05, and 1.94 cm ± 0.12. A significant increase was identified using the six-score model for VYG in animals with VYG scores of "A+O" higher than "A+" and "A+AD", with respective least-squares means of 3.75 ± 0.19, 3.20 ± 0.09, and 3.20 ± 0.08 ($P < 0.05$). When using the 4-score system, HCW was significantly lower for animals with scores of "A+" compared to those with non-abscessed livers. Hot carcass weight least-squares means for animals with no abscesses was 396 kg ± 2.63, and for those with severe abscesses was 391 kg ± 2.92 ($P < 0.05$).

In the second study, 1,492 beef on dairy heifers fed and harvested in Kansas had liver abscess scores and sire information. Nine models were utilized to estimate heritability, all with fixed effects of contemporary group, age in days, and number of bovine respiratory disease treatments. The contemporary group was a concatenation of kill lot and treatment. Models 1, 4, and 7 were from data sets with all sires represented but had liver abscess score represented as a continuous variable, a binary score indicating abscess presence, and a binary score indicating severe abscess ("A+") presence, respectively. Models 2, 5, and 8 followed the same respective scoring systems as Models 1, 4, and 7, but the data set only included heifers from sires with 10 or more progeny. Models 3, 6, and 9 followed the same respective scoring systems as Models 1, 4, and 7, but the data set only included heifers from sires with 100 to 200 progeny in the complete data set. Heritability estimates from a sire model for Models 1, 4, and 7 ranged from $4.26 \times 10^{-8}$ to $1.06 \times 10^{-7}$. Heritability estimates from a sire model for Models 2, 5, and 8 ranged from 4.90 x

$10^{-8}$ to 4.61 x $10^{-7}$. Heritability estimates from a sire model for Models 3, 6, and 9 ranged from 1.01 x $10^{-7}$ to 2.88 x $10^{-3}$. All estimates indicate no genetic component to liver abscess severity or incidence in this data set.

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# CHAPTER 1: INTRODUCTION

Since the 1890s, liver abscesses in ruminants have concerned livestock producers (McFadyean, 1891). Abscess incidence rates fluctuate based on management, breed, and sex (Nagaraja and Chengappa, 1998). The primary cause of liver abscesses is believed to be acidosis. Acidosis occurs in cattle when they are on a diet with increased levels of grain, leading to an increased rate of fermentation and volatile fatty acid production, which decreases the pH of the rumen (Nagaraja and Titgemeyer, 2007; Hernández et al., 2014). Rumenitis is a sequela to acidosis characterized by irritation of the ruminal epithelium caused by low pH. Ulcers on the rumen wall allow naturally occurring bacteria in the rumen, such as *Fusobacterium necrophorum* and *Trueperella pyogenes*, to enter the splanchnic circulatory system (Smith, 1944; Jensen et al., 1954; Nakajima et al., 1968). The splanchnic circulatory system carries nutrient filled blood from the gastrointestinal tract to the liver (Jennings and Premanandan, 2017). The bacteria enter the liver, and to prevent the spread of the bacteria, an abscess is formed (Nakajima et al., 1968). Historically, fed cattle and cull cattle have similar liver abscess rates, ranging from 18% to 20% (NBQA, 2016; Eastwood et al., 2017; Herrick et al., 2022), while fed Holstein cattle have a significantly higher incidence of liver abscesses at 25% (Herrick et al., 2022). Literature on fed beef on dairy has been reported as 50.18%, which was significantly higher than those for any other cattle type (Grimes, 2022). The effect of sex on incidence rate for fed cattle varies. Nagaraja et al. (1996) identified a significant increase in abscesses in steers; however, Herrick et al. (2022) identified a non-significant increase in abscesses in heifers.

Economic losses resulting from liver abscesses are most explicitly due to the required condemnation of the entire liver when any abscess is present. In severe cases where the liver has

adhered to nearby organs or there is contamination due to a ruptured abscess, additional condemnation of adhered and contaminated areas is required. The overall economic loss due to a liver condemnation rate of 18% for 26,984,200 slaughtered animals was determined to be $15.8 million, but the additional condemnation of adhered or contaminated tissue in 3.5% of the animals led to an additional $7.0 million loss (Brown and Lawrence, 2010). Varied results on the effects on carcass performance have been determined, with some undesirable effects being associated with increased liver abscess severity. Severe liver abscesses include those with scores of “A+”, “A+AD”, and “A+O”. Depending on the study, those scores may be represented separately or as one score. Of the six studies that investigated the effects of liver abscess severity on carcass performance in fed cattle, five identified at least one group of cattle that had significant decreases in hot carcass weight (Grimes, 2022; Rezac et al., 2014; Brown and Lawrence, 2010; Montgomery, 1985; Brink et al., 1990; Fox et al., 2009). Average daily gain was analyzed in three of the studies and found a significant decrease in two of the three for animals with severe liver abscesses (Rezac et al., 2014; Brink et al., 1990; Fox et al., 2009). Final body weight was analyzed in three studies, but only one identified a decrease in those with severe abscesses (Montgomery, 1985; Fox et al., 2009; Brown and Lawrence, 2010). Two studies looked at dressing percentage. Three studies looked at fat thickness and yield grade. Dressing percentage in both studies had a significant decrease with severe liver abscess scores. Fat thickness was significantly decreased in those from two studies, and yield grade was significantly decreased in two of the studies and a subset of animals in the third study (Montgomery, 1985; Brown and Lawrence, 2010; Grimes, 2022). Of two studies looking at rib eye area, kidney, pelvic, and heart fat percentage, and marbling score there were significant decreases with increased abscess severity for rib eye area in both. Only one of the studies

identified significant decreases in kidney, pelvic, and heart fat percentage and marbling score with increased abscess severity and the other study found no significant differences in kidney, pelvic, and heart fat percentage and both increases and decreases in marbling score (Grimes, 2022; Brown and Lawrence, 2010). Additionally, a significant increase was found in meat color (Brown and Lawrence, 2010).

Each of the five studies discussed in the previous paragraph had traits that were found to have a significant association with liver abscess scores based on animal breed, experimental group, and sample population. Brown and Lawrence (2010) further investigated the discounts and premiums associated with different liver abscess scores. While hot carcass weight was significantly decreased for the majority of the liver abscess scores compared to no abscess, only the discount for those with scores of "A+O" were significantly different animals with normal livers. The "A+O" discount was less than that of any other score, indicating an economic increase compared to carcasses with non-abscessed livers. Quality grade discounts were significantly worse for animals with scores of "A+AD" and "A+O". Yield grade had premiums for animals with severe scores and a lesser discount for those of "A-" compared to non-abscessed livers due to lower yield grades in animals with abscessed livers. Regardless of the premiums found in yield grade, animals with any liver abscess score had significantly lower gross carcass values than animals with non-abscessed livers (Brown and Lawrence, 2010). The loss of gross carcass value contributes to the total economic loss that occurs due to liver abscesses.

To combat economic losses due to liver abscesses, prevention methods have been used to decrease incidence rates. The prevention method of most use in conventional beef systems are antimicrobial feed additives. The additive with the highest efficiency is tylosin phosphate. When fed tylosin, incidence rates have been shown to decrease from 30% to 8%; however, tylosin does

not completely eradicate liver abscesses in those populations (Wileman et al., 2009). In addition to not being 100% successful in prevention, consumer perception of antibiotics has worsened, with many consumers concerned about antibiotic use in food animals (Barrett et al., 2021). Nutritional management remains a holistic prevention method, with diet formulation and management to decrease acidosis incidence (Owens et al., 1998). Acidosis can be successfully decreased in the herd, but improper feed management or focus on high grain diets to increase gain can make proper management difficult. Keele et al. (2016) began to move the focus in research to genetic components of liver abscesses in beef cattle. Genetic variance was identified for liver abscess using a genome-wide association that identified 35 single nucleotide polymorphisms associated with liver abscess incidence (Keele et al., 2016). With implications of genetic variance, a new prevention method of genetic selection may be available for producers.

While previous research efforts have investigated liver abscesses in beef cattle and Holstein cattle, beef on dairy cross cattle have yet to be investigated regarding genetic contribution. Therefore, using beef on dairy heifer information, the objectives of this study were to produce effect estimates for the impact of liver abscess scores on hot carcass weight, fat thickness, rib eye area, calculated visual yield grade, marbling score, and heart score and to produce preliminary heritability estimates for liver abscess severity and incidence.

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# CHAPTER 2: CATTLE LIVER ABSCESS REVIEW OF LITERATURE

## SECTION 1: LIVER PHYSIOLOGY IN MAMMALS

The liver of ruminant animals is a lobed glandular organ located adjacent and medial to the right body wall of the abdomen behind the diaphragm (Braun, 2009; Church, 1988). The majority of the organ is located on the right side of the body. The only segment of the liver located on the left side of the body is the portion of the left lobe that is ventral to the esophagus (Budras et al., 2003). The liver is approximately 1 to 1.3% of the animal's body weight and a reddish-brown color (Church, 1988). The liver is a part of the mid-gut and has a significant impact on the metabolic function of mammals as it plays a main exocrine role and a partial endocrine role (Macchiarelli et al., 1990).

The lobes of the liver can be described in two varying ways: functional and morphological. The morphological lobes are the right and left lobe. The right lobe is larger and further categorized into the caudate lobe and quadrate lobe. The caudate lobe is located along the inferior vena cava. The quadrate lobe is located between the gallbladder and round ligament (Kuntz and Kuntz, 2008). In ruminants, the distinction of right and left lobe is not evident macroscopically because it has less interstitial connective tissue (Church, 1988). While the distinction is not macroscopic, the falciform ligament is still present (Budras et al., 2003). There are nine functional segments of the liver based on portal vein flow. The segmentation is based on the Rex-Cantile line (Kuntz and Kuntz, 2008).

The liver is attached to the splanchnic circulatory system which makes up the blood flow portion of the intrahepatic vascular system. The hepatic artery brings oxygen-rich blood from the

heart to the liver (Jennings and Premanandan, 2017). The hepatic artery supplies blood to the peribiliary vascular plexus, portal fields interstitium, portal vein vasa vasorum, hepatic vein vasa vasorum, and the liver capsule (Kuntz and Kuntz, 2008). The portal vein brings blood into the liver that has passed through the gastrointestinal tract, gallbladder, spleen, and pancreas. The portal vein blood is poor in oxygen but does contain nutrients from the digestive tract (Jennings and Premanandan, 2017). The blood from the portal vein enters the sinusoid capillaries. Blood received from the portal vein makes up around 75%, while hepatic artery blood makes up the remaining 25% (Kuntz and Kuntz, 2008). High volumes of blood pass through the liver, with approximately 40 ml/minute/kg of body weight of blood passing through the portal vein (Huntington, 1990). The blood that entered the liver through the hepatic artery and portal vein, then leaves the liver through the hepatic veins back to the heart (Jennings and Premanandan, 2017). The superior hepatic veins drain into the inferior vena cava (Kuntz and Kuntz, 2008).

Most of the total cell numbers in the liver are hepatocytes, making up approximately 60% (Kuntz and Kuntz, 2008). Hepatocytes are the functional cells of the liver and are epithelial cells with a polygonal shape. The functions of hepatocytes include endocrine, exocrine, glucose storage, gluconeogenesis, deamination, and detoxification. They secrete plasma protein and bile, store glycogen, produce glucose and urea, and break down toxins (Mescher et al., 2021).

The remaining 40% of total cell numbers are made up of sinusoidal cells. The sinusoidal cells consist of endothelial cells, Kupffer cells, perisinusoidal cells, and PIT cells (Macchiarelli et al., 1990). The endothelial cells are the most prevalent sinusoidal cell type and are responsible for sinusoid capillary lining. These cells have a production and secretion role for cytokines, matrix components, growth factors, and vasoactive substances (Kuntz and Kuntz, 2008). The Kupffer cells are macrophages that adhere to the endothelial cells that make up the sinusoid

lining (Macchiarelli et al., 1990). In addition to their role as macrophages, Kupffer cells can also discharge the substances secreted by the endothelial cells, perform pinocytosis, and clear toxins, antigens, etc. (Kuntz and Kuntz, 2008). The perisinusoidal cells – also known as Ito cells, fat-storing cells, and stellate cells – have the primary role of storing cytoplasmic lipids, vitamin A, and fibrogenesis through the synthesis and secretion of collagen, fibronectin, etc. (Macchiarelli et al., 1990; Kuntz and Kuntz, 2008). The last type of sinusoidal cells are PIT cells. PIT cells are located near endothelial and Kupffer cells and are large granular lymphocytes. PIT cells are not fully understood but can play a large part in controlling liver disease by eradicating tumor cells, foreign cells, and necrosed cells (Macchiarelli et al., 1990; Kuntz and Kuntz, 2008).

## SECTION 2: LIVER FUNCTION IN RUMINANTS

The liver's functions are heavily related to metabolism. The liver plays a significant role in carbohydrate, protein, and lipid metabolism, being one of the most metabolically important organs in mammals. Outside of metabolism, the liver also has processes related to hormone synthesis and degradation, and detoxification. The process most important to carbohydrate metabolism is gluconeogenesis. The processes most important to protein metabolism are protein turnover and urea synthesis. The processes most important to lipid metabolism are ketogenesis, phospholipid and cholesterol synthesis, and the release of lipoproteins. Other processes important to lipid metabolism but specific to bile function are the synthesis and release of bile and the assimilation of bilirubin. Important processes related to hormones are the release and degradation of plasma proteins and peptide hormones. Another important process of the liver is detoxification (Danfær, 1994).

For ruminants, gluconeogenesis is a critical metabolic function. Ruminants do not absorb dietary glucose into the blood, so they need to utilize propionate, gluconeogenic amino acids, glycerol, and lactate to synthesize glucose. Glucose is necessary for the process of glycolysis, which produces adenosine triphosphate (ATP) that is utilized as cellular energy. Nervous tissue, muscle tissue, adipose tissue, mammary gland tissue, and fetal tissue all require glucose as their primary fuel source (Church, 1988). In the absence of glucose, the body will begin utilizing fat to produce ATP. When the animal is in a starving state, and there are no gluconeogenic substrates for the liver to utilize in gluconeogenesis, it will begin ketogenesis (Kohlmeier, 2003). When protein is consumed by the animal, the ruminant will gain access to the amino acids via undegradable protein or microbial protein. When the amino acids are absorbed from the small intestine, they enter the liver. In the liver, there are three routes that can take based on the energy status of the animal. The amino acids can exit the liver and enter the bloodstream, traveling to tissues that require protein or contribute to milk protein in the lactating female. The amino acids can also be transformed into plasma proteins, e.g. albumin. Finally, the amino acids may also be utilized for ATP production, either through direct oxidation or gluconeogenesis (Church, 1988). When utilized for ATP production, the amino acid is deaminized. The removed amine group produces ammonia and is converted to urea in the hepatocytes (Charlton, 1996). Urea is important for ruminant animals because of the need for nitrogen in the life cycle of ruminal microorganisms contributing to the production of microbial protein. If the urea levels are too high, the animal may also excrete the urea in the urine (Owens and Zinn, 1988).

As an exocrine gland, the liver secretes bile. The bile exits through the bile ducts, which empty into the hepatic duct and, subsequentially, the cystic duct and is stored in the gallbladder (Church, 1988). The synthesis of primary and tertiary bile acids occurs in hepatocytes.

Secondary bile acids are only biosynthesized in the intestines, and tertiary bile acids are formed in the intestines and liver. The biosynthesis of primary bile acid comes from the formation of cholesterol to cholic acid and chenodeoxycholic acid by 7α-hydroxylase. Secondary bile acids form when cholic acid and chenodeoxycholic acid enter the intestine and form deoxycholic acid, lithocholic acid, and ketolithocholic acid. Tertiary bile acid is formed when lithocholic acid enters the liver and is converted to sulpholithocholic acid, and ketolithocholic acid enters the liver and intestine and is converted into ursodeoxycholic acid. Bile in essential to the metabolism of cholesterol, digestion and reabsorption of dietary lipids, emulsification of fat-soluble vitamins, and stimulation of the intestine (Kuntz and Kuntz, 2008).

## SECTION 3: LIVER ABSCESS SCORING

A heavily utilized liver abscess scoring system is the Elanco Liver Check System (Elanco, Greenfield, IN). The liver abscesses are categorized as small or large, which measure less than 2.5 cm or greater than 2.5 cm, respectively. A score of "0" indicates a normal liver with no abscesses. A score of "A-" indicates a liver with 1 to 2 small abscesses. A score of "A" indicates a liver with multiple small abscesses or 1-2 large abscesses. A score of "A+" indicates a liver with multiple large abscesses. A score of "A+AD" indicates that the liver is adhered to the gut or diaphragm, regardless of liver abscess size. A score of "A+O" indicates that the liver abscess has ruptured, regardless of liver abscess size (Texas Tech University, 2023). A visual representation of the scoring system is illustrated in Figure 2.1. Scores of "A+AD", "A+O", and "A+" may be combined into a broader score of "A+" indicating severe liver abscesses. Only livers with a score of 0, or no presence of any abscesses, are edible and used for human

consumption. Any liver with any abscesses present, regardless of size or number, is condemned. Some may categorize the scores into normal and condemned rather than the four or six score system.

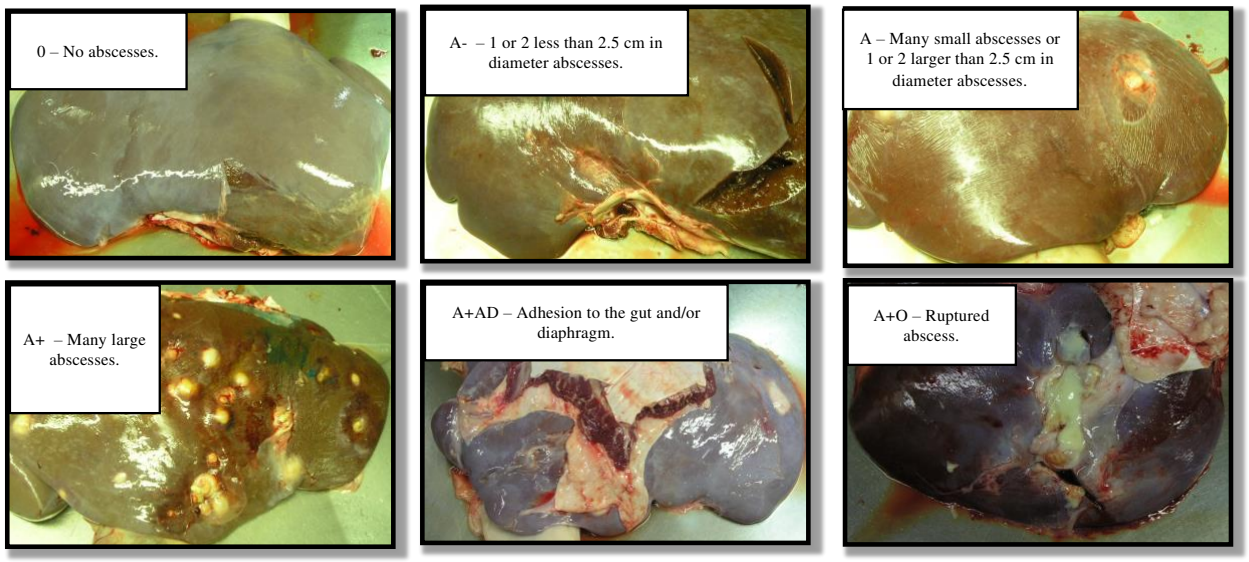


Figure 2.1 Visual representation and written descriptions of liver abscess scoring system provided by Texas Tech University.

## SECTION 4: PATHOGENESIS OF LIVER ABSCESSES IN CATTLE

The pathogenesis of liver abscesses differs between ruminants and non-ruminants. While non-ruminants form pyogenic liver abscesses due to biliary tract disease and appendicitis, ruminants form liver abscesses typically as sequela to ruminitis (Longworth and Han, 2015; Nagaraja et al., 2005). McFadyean (1891) hypothesized that the cause of the liver abscesses was inflammation of the intestine allowing the causative agent to enter the portal system. The leading hypothesis for what caused liver abscesses in the late 1930s was that it was due to higher feeding of beet by-product, soybean meal, or the presence of a foreign body (Newsom, 1938; Frederick, 1943). Only six years later, the theory relating ruminitis – a disorder characterized by

inflammation and ulceration of the rumen – to liver abscesses that was originally hypothesized in the 1890s was established (Smith, 1944). Smith (1944) tested this hypothesis by evaluating the liver and rumen of 1,807 slaughtered beef cattle. The study identified any lesions on the rumen wall and any abscesses in the liver. The findings revealed that of the 474 animals with ruminal lesions, 42% also had liver abscesses. Of the 332 animals with liver abscesses, 62% had ruminal lesions. Conversely, of the 1,333 animals without ruminal lesions, only 9% had liver abscesses. Of the 1,485 animals without liver abscesses, only 18% had ruminal lesions. The percentage of animals having both liver abscesses and ruminal lesions being higher than those without one or the other led to the conclusion that there is a connection between ruminal lesions and liver abscesses. The connection between the rumen and the liver through the portal vein helped form the theory (Smith, 1944).

A decade after the publication of Smith (1944), Jensen et al. (1954b) utilized inoculation of ruminant animals to gather more information about the pathogenesis of liver abscesses in cattle and sheep. The inoculation protocol was done via the portal vein, which refers back to the idea from Smith (1944) that the portal vein may be the method of metastasis from the ruminal lesions. Following inoculation, 69.7% of the cattle had liver abscesses, and 90% of the sheep had liver abscesses. The necropsy of the animals was done between 1.5 days to 183 days post-inoculation. The inoculation was done using the bacteria *Spherophorus necrophorus*, more currently referred to as *Fusobacterium necrophorum.* The presence of *F. necrophorus* in the liver abscesses confirmed the belief that *F. necrophorus* was a causative agent (Jensen et al., 1954b). Jensen et al. (1954a) in the same year published a study that formally defined the rumenitis-liver abscess complex.

Wieser et al. (1966) did not find the same correlation between rumenitis and liver abscesses but instead a correlation between rumenitis and fed cattle. Rezac et al. (2014) found in their study of beef cattle that 32% of the cattle with rumenitis had a liver abscess, but 19% of the cattle without rumenitis had a liver abscess. The proportionality of those with rumenitis and liver abscesses was not as intense as that of Jensen et al. (1954b), but the authors did note that there were potentially experimental components that contributed to the differences. Ruminitis itself is a sequela to acidosis. Acidosis is a disorder of the rumen that is caused by the accumulation of organic acids (Nagaraja and Titgemeyer, 2007). The main causes of acidosis in fed cattle are high grain consumption, inadequate ruminal buffering, and poor bunk management (Hernández et al., 2014). High-concentrate diets are the diet type most common in feedlots and dairy operations.

In the digestive system of the ruminant, mastication in the mouth occurs first. Unlike monogastric animals, the ruminant animal does not contain salivary amylase and lipase in the saliva to begin chemical digestion of feed (Molnar and Gair, 2015). The saliva of the ruminant does contain bicarbonate and phosphate, which acts as a buffer for the rumen to maintain a neutral pH (Church, 1988). When fed a high-concentrate diet, the grains are nonstructural and easier for the microorganisms of the rumen to ferment without a need for extensive mastication. The decreased time of mastication also decreases saliva production, which then decreases bicarbonate and phosphate levels entering the rumen (Hernández et al., 2014).

After the feed is swallowed by the animal, it enters the reticulorumen. In the reticulum, denser feed remains to be regurgitated for continued rumination. Then the ingesta enters the rumen. In the rumen of a mature ruminant, there are numerous categories of microorganisms responsible for fermentation, including bacteria, protozoa, fungi, mycoplasma, and bacteriophages (Church, 1988). The microorganisms responsible for the fermentation of

carbohydrates are amylolytic bacteria (Hernández et al., 2014). In the anaerobic environment of the rumen, the bacteria go through glycolysis to produce phosphoenolpyruvate (PEP), which by the enzyme pyruvate kinase is transformed into pyruvate (Weimer and Kohn, 2016; Kennelly et al., 2023). The pyruvate is then utilized in the synthesis of volatile fatty acids (VFA), also known as short chain fatty acids (SCFA). The VFA of particular importance in the rumen are acetate, propionate, and butyrate which are 2, 3, and 4 carbons in length, respectively. Moss et al. (2000) summarizes the fermentation pathways as:

$$Pyruvate + H_2O \rightarrow Acetate\ (C_2) + CO_2 + 2H$$

$$Pyruvate + 4H \rightarrow Propionate\ (C_3) + H_2O$$

$$2C_2 + 4H \rightarrow Butyrate\ (C_4) + 2H_2O$$

The fermentation of the grain is rapid due to the decreased particle size and increased surface area. The increased production of VFA at a rapid pace is higher than the absorption rate of the VFA into the blood, causing an accumulation of VFA in the blood (Owens et al., 1998). The accumulation of VFA in the rumen is typically exacerbated by rumenitis or abnormal ruminal papillae, decreasing absorption (Nagaraja and Titgemeyer, 2007). When the rumen is experiencing an accumulation of VFA, the pH of the rumen will decrease. Ideally, the rumen maintains a pH of 5.5 to 6.5. The $pK_a$ of VFA is around 4.8, so when the pH is above 4.8, the VFA is dissociated, causing a decrease in pH (Nagaraja and Titgemeyer, 2007; Hernández et al., 2014). If the pH is below the $pK_a$ value, then the VFA becomes protonated, removing hydrogen ions from the environment. A decrease in hydrogen ions present in the solution is associated with an increase in pH, helping the pH return to neutral (Brandt et al., 2017). This increase in pH benefits the ruminant animal by allowing absorption of VFA to resume and increase in rate, acting as a buffer to attempt to maintain homeostasis (Nagaraja and Titgemeyer, 2007; Owens et

al., 1998). However, this only functions when the pH decreases to below 4.8. Before that occurs, the pH is still below the normal of 5.5. This drop in pH is indicative of microbiome changes in the rumen.

In response to the acidic pH, sensitive bacteria, mainly gram-negative bacteria and lactate-consuming bacteria, will die. When the gram-negative bacteria die, there is an indication that they produce lipopolysaccharides, also known as bacterial endotoxins. The bacteria resistant to the drop in pH, such as some gram-positive bacteria and lactate-producing bacteria, will survive. The lactate-producing bacteria, such as *Streptococcus bovis*, are producing L-lactic acid in the rumen. The L-lactic acid has a lower $pK_a$ than VFA, being 3.8 compared to 4.8, respectively. This lower $pK_a$ value indicates that lactic acid is a stronger acid than VFA, dissociating more hydrogen ions and, subsequently, having a larger impact on the pH decrease (Owens et al., 1998; Nagaraja and Titgemeyer, 2007; Hernández et al., 2014; Monteiro and Faciola, 2020). This secondary pH decrease continues to kill off any acid sensitive bacteria, leaving lactate-producing bacteria, especially *Lactobacilli* subsp., behind to continue to produce lactic acid and continue the pH decrease (Hernández et al., 2014). The acidic pH of the rumen also causes the osmolality of the rumen contents to increase, decreasing the rate of absorption of acids (Owens et al., 1998). The acidic rumen environment is indicative of acidosis. Eventually, when the ruminal pH reaches the $pK_a$ value of lactic acid – 3.8 – the acids will become protonated and be absorbed into the bloodstream, causing metabolic acidosis. The previously mentioned decrease in saliva production due to decreased mastication required for nonstructural carbohydrates also aggravates acidosis development due to the decrease of buffer to help maintain the neutral pH (Hernández et al., 2014).

The severity of the ruminal acidosis is dependent on the pH level. Subacute acidosis is less severe and asymptomatic. The pH is slightly below normal, ranging from 5.0 to 5.5 and more likely caused by increased VFA production and decreased absorption, associated more with the first microbiome population shift and beginning decrease of pH. Acute acidosis is symptomatic and more severe. The pH is significantly below normal, being below 5.0. It is caused by the increase in lactic acid production, associated more with the second microbiome population shift. While severity does differ, rumenitis and liver abscesses can be caused by subacute or acute acidosis (Nagaraja and Titgemeyer, 2007).

"True stomachs", such as the stomach in the monogastric and the abomasum in the ruminant, are acidic due to its function in chemical digestion. The pH of these stomachs are between 1.5 and 2.5, much lower than that of a healthy rumen (Molnar and Gair, 2015; Jennings and Premanandan, 2017). The stomach has a mucosal barrier to protect the stomach epithelium from the acidic gastric juice. The mucus is alkaline, which acts as a buffer against the low pH of its environment. The presence of mucus on the stomach wall is what allows the stomach to maintain such a low pH while preventing damage to the tissues of the stomach (Merchen, 1988). The rumen epithelium does not have the same type of mucus lining because the rumen is not intended to be an acidic environment, so injury to the epithelium can occur when the animal has acidosis. The ruminal epithelium can get inflamed, erode, form ulcers, or develop parakeratosis. If the epithelium is inflamed, eroded, or ulcered, the bacteria present in the rumen – and their endotoxins – may enter the splanchnic circulatory system (Krause and Oetzel, 2006; Oetzel, 2017; Aschenbach et al., 2019; Plaizier et al., 2018). While the VFA stimulate ruminal papillae growth, the high levels of VFA in an acidic environment disrupt the normal papillary growth, leading to the development of parakeratosis in the rumen. Parakeratosis contributes to rumenitis,

even having the potential to form micro-abscesses that allow for the translocation of bacteria and bacterial endotoxins. In addition to the abscessation of the rumen, parakeratosis and the established rumenitis-liver abscess complex may contribute to the decrease in performance seen in cattle with liver abscesses (Hernández et al., 2014; Montgomery, 1985; Brown and Lawrence, 2010; Brink et al., 1990; Fox et al., 2009).

While the primary cause of liver abscesses is identified as rumenitis, there is the ability for liver abscesses to be caused by traumatic reticuloperitonitis or injury to the hepatic artery or biliary tree. The neonate may also be susceptible to liver abscesses through injury to the umbilical cord. Rumenitis may be exacerbated by rough and sharp ingesta (Kelly, 1993).

The timing of liver abscess formation was reported by Nagaraja and Lechtenberg (2007), with personal findings of the last sixty days of the feeding period being when the liver abscesses primarily develop, with ones developing at the beginning of the feeding period typically being healed within 2 months. The severe liver abscesses found during slaughter followed the trend of developing within the last sixty days (Nagaraja and Lechtenberg, 2007).

The connection of liver abscesses as a sequela to rumenitis is due to the ability of bacteria to be translocated into the portal blood flow because of the damaged ruminal epithelium. When the bacteria enter the bloodstream from the rumen, due to the pathway of the splanchnic circulatory, the first organ they will enter is the liver (Drackley et al., 2006; Amachawadi and Nagaraja, 2016). Prior to experimentation in cattle, it was done on a smaller mouse model by Abe et al. (1976). In mice, when infected with *F. necrophorum*, they began to exhibit signs of infection three days post-inoculation. Three to thirteen days post-inoculation, animals were necropsied, revealing abscesses primarily in the liver. Coagulative necrosis and micro-abscessation was found from the three to five-day post-inoculation range. Ten days post-

inoculation, the livers had abscesses with pus and necrotic tissue. Enclosing the abscess cavity was necrotic polymorphonuclear leukocytes, abundant fibrinous tissue, and partially necrotic parenchymal tissue. Operational hepatocytes and leukocytes were at the distal ends of the capsule. Kupffer cells were multivacuolated and enlarged, hepatic sinusoids contained small aggregates of mononuclear cells, and hepatocytes outside of the abscessed area were not affected (Abe et al., 1976).

Nakajima et al. (1986) performed an experimental infection study for cattle. The findings revealed similar time frames as seen in mice from the Abe et al. (1976) study, with prior to 5 days identifying coagulative necrosis and micro-abscesses and after 9 days seeing the presence of developed abscesses. The Nakajima et al. (1986) study does begin an investigation of the liver closer to inoculation. At 12 hours post-inoculation, the liver was spread with white foci of 0.5 to 1 mm in diameter. These were in the sinusoids, contained eosinophilic substances, and had hepatocyte involvement. The necrotic foci and sinusoids had hemorrhages and microthrombi of fibrin in and around them. From 24 to 36 hours post-inoculation, the liver had numerous foci that were a whitish-yellow and 2 to 3 mm in diameter. From 2 to 4 days post-inoculation, the foci were now 1 cm in diameter, were caseous with an irregular round shape. If near the surface, they were slightly elevated. For all 1.5 to 4 days post-inoculation, they had congestions or hemorrhoids, cloudy swelling, nuclear debris, bacteria, and homogenous tissue mass in the center, shrunken hepatocytes, and indication of inflammatory response. They also had been enlarged and often were fused. For 9 or more days post-inoculation, the livers had 3 to 10 abscesses of diameters ranging from 0.6 to 10 cm. The tissues were caseous and contained pus and granulation. There was coagulative necrosis, nuclear debris, and the capsule has fibrinous exudates, plasma cells, and fibrous tissue (Nakajima et al., 1986). Jensen et al. (1954b) found

similar results, but also produced data regarding the healing of the abscesses. In the range of 46 to 183 days, scars began to form. The adhesion reported in the scoring of A+AD was observed to be due to fibrinous inflammation of abscesses present on the surface of the liver (Jensen et al., 1954b). The fibrinous inflammation caused the adhesion to the organs near the liver, such as the peritoneum, diaphragm, and gastrointestinal tract (Nagaraja and Lechtenberg, 2007).

## SECTION 5: BACTERIA ASSOCIATED WITH LIVER ABSCESSES

There are numerous bacteria that have been identified in liver abscesses. Anaerobic and aerobic bacteria can be present in liver abscesses; however, anaerobic bacteria are most prevalent (Lechtenberg et al., 1988; Nagaraja and Chengappa, 1998). The primary causative agent identified is *Fusobacterium necrophorum,* previously named *Spherophorus necrophorus* and *Actinomyces necrophorus.* The second most common bacteria identified is *Trueperella pyogenes*, previously named *Corynebacterium pyogenes, Actinomyces pyogenes,* and *Arcanobacterium pyogenes* (Schoch et al., 2020). Newsom (1938) found that most abscesses sampled from the slaughtered cattle had *F. necrophorum*, with *T. pyogenes* also being found occasionally. This trend continues to be observed in numerous studies over the decades (Nagaraja and Chengappa, 1998). Other less-common bacteria isolated include *Bacteroides* subsp., *Clostridium* subsp., *Escherichia coli, Klebsiella* subsp., *Enterobacter* subsp., *Mobilincus* subsp., *Pasteurella* subsp., *Peptostreptococcus* subsp., *Porphyromonas* subsp., *Prevotella* subsp., *Propionibacterium* subsp., *Staphylococcus* subsp., and *Streptococcus* subsp. Unidentified bacteria have also been isolated from liver abscesses (Amachawadi and Nagaraja, 2016). A prevalence breakdown was provided in a study by Nagaraja and Lechtenberg (2007), revealing the prevalence of *F. necrophorum*

found in liver abscesses was 100% – 71% having *F. necrophorum* subsp. *necrophorum* and 41% having *F. necrophorum* subsp. *funduliforme*. The prevalence of *T. pyogenes* was 32% and 49% contained other bacteria. This ranking is consistent with that of Purvis (2006) and is consistent for both liver abscesses secondary to rumenitis and liver abscesses secondary to traumatic reticuloperitonitis. In order of highest to lowest prevalence it was *F. necrophorum* – subsp. *necrophorum* then subsp. *funduliforme –, T. pyogenes,* and *Clostridium perfringens* (Amachawadi and Nagaraja, 2016). A more recent study by Herrick et al. (2022) analyzed liver abscesses in seven fed-beef processing plants and four cull-beef processing plants from 2015 to 2016. Of all fed-beef processors, *F. necrophorum* was present in all abscesses, with *F. necrophorum* subsp. *necrophorum* having the highest prevalence of all bacteria isolated (79.9%). *F. necrophorum* subsp. *funduliforme* had the third highest prevalence (24.3%), *Salmonella enterica* being the second most prevalent (27.5%), and *T. pyogenes* having the least prevalence (14.8%). Of the seven fed-beef processing plants, four had prevalence of *Salmonella enterica,* three had prevalence of *T. pyogenes,* and one had prevalence of *T. pyogenes* and *Salmonella enterica.* The cull-beef processing plants had a slightly different incidence ranking, with *F. necrophorum* subsp. *necrophorum* being first (76.9%), then *F. necrophorum* subsp. *funduliforme* being second (17.6%), then *Salmonella enterica* third (16.5%), and *T. pyogenes* fourth (8.8%). Of the four individual plants, only one had prevalence of *Salmonella enterica,* three had prevalence of *T. pyogenes,* and one had prevalence of both *Salmonella enterica* and *T. pyogenes* (Herrick et al., 2022). While the *F. necrophorum* subspecies and *T. pyogenes* remain consistent with previous reports, the presence of *Salmonella enterica* is a more recent development, the first isolation from liver abscesses being found in 2013 and reported in 2015 (Amachawadi and

Nagaraja, 2015). The new introduction of *Salmonella enterica* in feedlot liver abscesses is not fully studied and the pathogenesis of it is unknown (Herrick et al., 2022).

*F. necrophorum* is a gram-negative bacterium that is rod-shaped and anaerobic. It does not form spores and is able to produce propionic acid from lactic acid (Nagaraja et al., 2005). The biotypes of *F. necrophorum* are A, B, AB, and C. The biotypes A and B are most isolated from liver abscesses, AB is rarely isolated, and C is nonpathogenic. Biotype A is the predominant isolate from liver abscesses. In an experimental study by Lechtenberg et al. (1988), *F. necrophorum* was isolated from all liver abscesses, with 57% having biotype A isolated from the abscess and 47% having biotype B isolated from the abscess. Biotype A has slightly less glucose and fructose fermentation than biotype B. Biotype A is more pathogenic than biotype B. Both biotypes produced increased propionate and decreased butyrate in the presence of lactic acid. The severity of lesions decreased when the pure culture of the abscess had biotype B (Lechtenberg et al., 1988). Biotype A is also known as *F. necrophorum* subsp. *necrophorum* and biotype B is also known as *F. necrophorum* subsp. *funduliforme* (Nagaraja et al., 2005).

The virulence factors of *F. necrophorum* – as condensed by Nagaraja et al. (2005) – are the leukotoxin, endotoxin, hemolysin, hemagglutinin, adhesins (Pili), dermonecrotic toxin, platelet aggregation factor, and proteases. The leukotoxin contributes to the virulence by protecting against neutrophil and Kupffer cell phagocytosis due to it being cytotoxic. It releases cytolytic products to damage the hepatic parenchyma and is considered the main virulence factor. The endotoxin contributes to virulence because it creates an anaerobic microenvironment due to its necrotic effect and dissemination of intravascular coagulation. The hemolysin contributes to virulence by getting iron from the host to support bacterial growth by lysing erythrocytes and helps contribute to the anaerobic environment. The hemagglutinin agglutinates erythrocytes

which assist in the attachment to the epithelial cells of the rumen and the hepatocytes. The adhesins (Pili) attach to the eukaryotic cell surface, which assists in the colonization of the ruminal epithelium or skin of the animal. The dermonecrotic toxin helps penetrate the ruminal epithelium or skin of the animal by causing epithelium necrosis. The platelet aggregation factor continues the creation of an anaerobic micro-environment, and deposits fibrin to protect the bacteria by breaking down the cell protein. The proteases also break down cell protein and contribute to the penetration of the ruminal epithelium or skin of the animal (Nagaraja et al., 2005; Tadepalli et al., 2009). All virulence factors contribute to the development of liver abscesses. The breakdown and eventual entry through the rumen wall from hemagglutinin, adhesins, dermonecrotic toxin, and protease activity allow access into the portal vein and, subsequently, the liver. In the liver, the leukotoxin, endotoxin, hemolysin, and platelet aggregation factor activity contribute to the establishment of an anaerobic environment, allowing the bacteria to survive and avoid phagocytosis in the liver.

With the established rumenitis-liver abscess complex and the identified primary causative agent being *F. necrophorum,* an interest in the location of *F. necrophorum* arose. Narayanan et al. (1997) looked to compare the *F. necrophorum* present in the liver abscesses and that naturally occurring in the rumen and on the rumen wall. The samples taken of *F. necrophorum* were categorized into the subspecies of subsp. *necrophorum* and subsp. *funduliforme.* Subsp. *necrophorum* was most isolated in liver abscesses, then ruminal contents, then ruminal walls. Subsp. *funduliforme* was most isolates in liver abscesses, then ruminal walls, then ruminal content. For both subspecies, the leukotoxin titer was significantly less in ruminal contents than it was in ruminal walls or liver abscesses. There was no significant difference between leukotoxin concentration (μg/ml), hemolysin titer, or hemagglutination titer. When ribotyping the

isolates, any results with one or more bands differing were considered distinct strains. All *F. necrophorum* isolates had bands at 20, 3.8, 3.6, and 3.4 kb. Subsp. *funduliforme* had an additional band at 4.3 kb. The subsp. *necrophorum* present in the ruminal content did not have a band at 2.4 kb, even though all others did and were the only ones to have bands at 6.0 and 3.3 kb. Isolates of two different liver abscesses from the same animal were identical in 83% of the animals. Isolates from liver abscesses and from the ruminal wall were identical for 89% of the animals. No isolates from the ruminal content were identical to that of the ruminal wall or liver abscesses from the same animal. The ruminal content may have differing strains of *F. necrophorum* due to a larger population and increased function. However, the ruminal wall and liver abscess isolates being identical may imply that there are fewer strains that attach to the ruminal wall. The strains that do attach to the ruminal wall seem to have higher leukotoxicity, potentially indicative of high virulence and that is how they then enter the liver to form abscesses (Narayanan et al., 1997). The study supported the hypothesis that the *F. necrophorum* causing liver abscesses comes from the rumen, but more specifically supports that it comes from that living on the rumen wall.

*F. necrophorum* is not only the pathogenic agent in bovine liver abscesses but has pathogenic agents for other diseases in cattle and various species, specifically necrobacillosis diseases. Foot rot and foot abscesses in cattle and sheep are associated with *F. necrophorum* infections, both subsp. *necrophorum* and subsp. *funduliforme* being isolated. Necrotic laryngitis in calves is associated with *F. necrophorum* infection, both subsp. *necrophorum* and subsp. *funduliforme* being isolated. Summer mastitis in cows is associated with *F. necrophorum.* Mandibular abscesses in antelopes and marsupials are associated with *F. necrophorum,* both subsp. *necrophorum* and subsp. *funduliforme* being isolated. In humans, Lemierre's syndrome

and abscesses in soft tissue are associated with *F. necrophorum*, the association specifically with subsp. *funduliforme* is undetermined. All diseases identified, except for those in humans, are also commonly associated with *T. pyogenes* (Nagaraja et al., 2005).

*T. pyogenes* is a gram-positive bacterium that can be coccobacillary or short rods. It does not form spores and are facultative anaerobes. *T. pyogenes* produces lactic acid from the fermentation of proteins and sugars. The virulence factors of *T. pyogenes* are cell wall macromolecules, pyolysin O, neuraminidases, extracellular matrix-binding proteins, fimbriae, proteases and other enzymes, and biofilm. Cell wall macromolecules cause the release of pro-inflammatory cytokines when interacting with macrophage cells. Pyolysin O is a hemolytic exotoxin that is active against various species of erythrocytes and is cytotoxic. It is stable in oxygen and is considered the primary virulence factor. Neuraminidases help in adhesion to the cells of the host by cleaving the terminal sialic acid from carbohydrates and from the host cell by cleaving the glycoproteins from their receptors. Extracellular matrix-binding proteins help in the adhesion to the cells of the host by binding to the extracellular matrix of the host. Fimbriae are not as thoroughly known but are thought to also contribute to adherence to the host cells. Proteases and other enzymes are theorized to support the decrease of immunity from the host and assist in invasion and destruction of host tissue (Nagaraja, 2022). The virulence factors of Pyolysin O, neuraminidases, extracellular matrix-binding proteins, and fimbriae all help the bacteria adhere to the host cell, while proteases and cell wall macromolecules allow for evasion of host immunity and tissue destruction.

*T. pyogenes* is associated with diseases other than liver abscesses. Metritis, endometritis, and mastitis are associated with *T. pyogenes* in cows and heifers, commonly found in combination with *F. necrophorum.* Foot rot, foot abscesses, and liver abscesses are associated

with *T. pyogenes* in cattle, commonly found in combination with *F. necrophorum*. Sheep and goats experience *T. pyogenes* associated disorders similar to that in cattle (Nagaraja, 2022). In white-tailed deer in the United States and Canada, *T. pyogenes* is associated with abscesses and lesions around the body and causes lumpy jaw and suppurative meningoencephalitis complex (Pothier et al., 2016; Nagaraja, 2022). In swine, *T. pyogenes* is associated with abscesses in organs that leads to systemic infections. Swine also experience *T. pyogenes* in association with respiratory, cardiac, skeletal, reproductive, and blood disorders. All swine disorders associated with *T. pyogenes* are polymicrobial. Common domesticated animals rarely experienced diseases associated with *T. pyogenes.* Humans may develop abscesses due to *T. pyogenes* infection, yet it is rare and mostly prevalent in those who are immunocompromised and exposed to livestock animals (Nagaraja, 2022). Overall, the diseases of *T. pyogenes* are most common to ruminants and livestock, typically cause abscesses, and are polymicrobial infections.

Narayanan et al. (1998) conducted a study to compare the *T. pyogenes* present in liver abscesses to those present in ruminal content and on the ruminal wall. Due to *T. pyogenes* not having subspecies like *F. necrophorum* does, the study also looked at *T. pyogenes*-like organisms to gain more information on the ribotyping and virulence factors of both. The ruminal wall and liver abscess isolates matched for two of six animals, one testing *T. pyogenes* and one testing *T. pyogenes*-like organisms. The ruminal content and liver abscess isolates matched for one of the two animals tested, specifically for *T. pyogenes*-like organisms. For *T. pyogenes*-like organisms both isolates taken from the rumen content and rumen wall matched. The ruminal content and ruminal wall had similarities to that of liver abscesses (Narayanan et al., 1998). In comparison to the study done on *F. necrophorum*, *T. pyogenes* isolates were similar for ruminal wall and

ruminal content, while *F. necrophorum* isolates were similar only for ruminal wall (Narayanan et al., 1997; Narayanan et al., 1998).

The impact of the bacterial species isolated from the liver abscesses and their corresponding liver abscess score was examined by Herrick et al. (2022). For samples with a liver score of A-, the rank of prevalence is: *F. necrophorum* subsp. *necrophorum* (71.8%), *Salmonella enterica* (20.5%), *F. necrophorum* subsp. *funduliforme* (10.3%), *T. pyogenes* (9.0%). *Salmonella* serotypes were also analyzed, with no isolation of Kentucky, Reading, or Schwarzengrund, and the ranking of prevalence for present serotypes was Anatum (9.0%), Montevideo (7.5%), Lubbock (4.5%), Mbandaka and Give both with the lowest prevalence (1.5%). For samples with a liver score of A, the rank of prevalence is: *F. necrophorum* subsp. *necrophorum* (62.2%), *F. necrophorum* subsp. *funduliforme* (24.4%), *Salmonella enterica* (23.3%), *T. pyogenes* (13.3%). The *Salmonella* serotypes of Give, Mbandaka, and Reading were not isolated, and the ranking of prevalence for present serotypes was Lubbock (11.9%), Montevideo (10.4%), Anatum (7.5%), Kentucky and Schwarzengrund both with the lowest prevalence (1.5%). For samples with a liver score of A+, the rank of prevalence is: *F. necrophorum* subsp. *necrophorum* (100%), *F. necrophorum* subsp. *funduliforme* and *Salmonella enterica* tied (27.7%), *T. pyogenes* (13.9%). The *Salmonella* serotype of Schwarzengrund was not isolated, and the ranking of prevalence for present serotypes was Anatum (13.4%), Montevideo (9.0%), Lubbock (7.5%), Kentucky (6.0%), Give and Reading (3.0%), Mbandaka (1.5%). The differences amongst liver scores were that as A- increased to A, *T. pyogenes* incidence increased, A+ had the highest incidence of *F. necrophorum* subsp. *necrophorum* and *Salmonella enterica*, and as severity increased, so did bacteriological incidence. The author indicates that the *Salmonella* serotypes identification and there being numerous types and

isolates identified may be due to the large geographical spread of the study across the United States (Herrick et al., 2022).

Lechtenberg et al. (1993) performed an experimental inoculation of *F. necrophorum, T. pyogenes,* and a combination to better understand the impacts of dosage on pathogenesis. Inoculation of pure *F. necrophorum* resulted in four of the five animals developing liver abscesses, and the isolates from those liver abscesses all being *F. necrophorum*. The inoculation of pure *T. pyogenes* did not result in any liver abscess formation at any point. The inoculation of a mixture of *F. necrophorum* and *T. pyogenes* did not result in any liver abscess formation at any point. However, the inoculation of a mixture of *T. pyogenes* with the leukotoxin of *F. necrophorum* did result in two of the five animals developing liver abscesses. The isolates from those liver abscesses were only identified as *T. pyogenes* (Lechtenberg et al., 1993). Liver abscess formation only occurring in the presence of *T. pyogenes* when it is combined with the leukotoxin of *F. necrophorum* may indicate the need for the leukotoxin specifically to form abscesses in the liver and that *T. pyogenes* may not increase the virulence of *F. necrophorum.*

## SECTION 6: ECONOMIC IMPACTS OF LIVER ABSCESSES

Liver abscesses have been of note in the beef industry since the formal definition was produced in the late 1800s (Jensen et al., 1954a; McFadyean, 1891). Reports of livers condemned for having abscesses in 1937 ranged from 1.88% to 10.37%, depending on the physical location of the facility (Newsom, 1938). The condemnation percentage for liver abscesses in fed cattle was 17.8% in 2016, a 4.1 percentage point increase from 2011 (Eastwood

et al., 2017). For cull cattle, there was a change in liver abscess prevalence from 13.7% in 2007 to 20.7% in 2016 (National Beef Quality Audit, 2016). The liver abscess rates are similar between fed and cull cattle in the National Beef Quality Audit, with market cows and bulls being 2.9 percentage points higher than fed cattle in 2016. Reported fed beef on dairy incidences are as high as 50.18% in a study of the animals in the West Texas Beef A&M University Beef Carcass Research Center, which was significantly higher than the reported incidences in Native, Holstein, and Mexican cattle from the study with respective rates of 23.02%, 39.24%, and 16.81% (Grimes, 2022).

Rates of liver abscesses and liver condemnation relate to profit loss. A study from 1942 reveals the profit loss due to liver abscesses from 1935 to 1942 to be $10,288,639.20 based on 3,674,514 condemned livers with an average wholesale value of $0.28 per liver (Frederick, 1943). That is approximately $1,469,805.60 annually. A study from 2010 reported an annual loss of $15,873,456.00 from liver abscesses, with an 18.1% incidence rate and an average wholesale value of $3.25 per liver in 2009. The 18.1% incidence rate is equivalent to 4,884,140 condemned livers based on their annual average of 26,984,200 slaughtered cattle. In addition to the loss from liver abscesses, those with adherence to the gut or ruptured abscesses produce an additional $7,007,797.00 annual loss with a 3.5% incidence and $7.42 per liver loss. Using the previous annual slaughter numbers, the 3.5% incidence is equivalent to approximately 944,447 condemned gastrointestinal tracts. The additional loss due to adherence to the gut or a ruptured abscess is because the processor must condemn the entire gastrointestinal tract, not just the liver (Brown and Lawrence, 2010). Herrick et al. (2022) estimated the economic impacts of liver abscesses from 2015 to 2016 based on their study. The study included data from fed-beef and cull-beef processing plants across the United States. The economic loss due to liver abscesses

from liver abscesses in fed-beef was $2.05/animal, with a total annual loss of $46.1 million (Herrick et al., 2022).

Montgomery (1985) was the first to formally study the potential for performance depression due to liver abscesses, even though it was suspected informally prior. The study does an analysis of all cattle slaughtered in the project, but has further separation into Holstein and non-Holstein, emphasizing any potential differences based on breed. This study fit scores of "A+", "A+AD", and "A+O" into one score for severe abscesses. For live weight and hot carcass weight (HCW), the all-cattle category and Holstein had no significant differences, but non-Holsteins showed animals with "A+" having a significant decrease. For dressing percentage, Holstein and non-Holstein showed a significant decrease in "A+", but the all-cattle category had significant decreases for all abnormal liver scores, decreasing as the scores became more severe. For fat thickness, Holstein and non-Holstein had no significant differences, but the all-cattle category showed "A+" having a significant decrease. For yield grade (YG), the all-cattle category and Holstein were significantly less for "A" and "A+", but non-Holstein had no significant differences. For quality grade (QG), Holstein and non-Holstein had no scores with significant differences, but the all-cattle category had "A+" having a significant decrease. The gut table trim percentage for all three breed categories showed "A+" having a significant increase. A significant increase was seen in the railout trim percentage for the all-cattle category and non-Holsteins with "A+" and Holstein with "A". A significant rise in the total trim due to abscesses percentage was seen in the all-cattle category and Holsteins showed that "A" and "A+" and non-Holstein with "A+" (Montgomery, 1985). A notable portion of the experimental design was that there were less Holstein cattle, with the sample size being in the 400s compared to non-

Holstein and the all-cattle category that had sample sizes over 1,000. The size discrepancy is likely due to prevalence of fed Holstein cattle being lower than non-Holstein cattle.

Brown and Lawrence (2010) studied the effects of liver abscesses on carcass traits and pricing. For the database without any anaphylactic treatments, they looked at body weight, dressing percentage, and HCW. There were no statistically significant differences in body weight. Dressing percentage had a statistically significant decrease in dressed yield percentage for those with scores of "A-", "A+", "A+AD", and "A+O". Hot carcass weight only had a significant difference from normal for those with a score of "A+AD". For the database that included treatment, they looked at carcass traits and economic impacts. For HCW, rib eye area, and kidney, pelvic, and heart fat percentage (KPH), "A-", "A+", "A+AD", and "A+O" were significantly lower than that of a normal liver. For USDA calculated YG and marbling score, "A+AD" and "A+O" were significantly lower than that of a normal liver. For $12^{th}$-rib subcutaneous fat depth, all abnormal liver scores were significantly lower. For color score, "A+" and "A+AD" were significantly higher than that of animals with a normal liver. Rib eye area/hot carcass weight and color score are the only carcass quality traits found in this study where any abnormal liver score had significantly higher scores. The high rib eye area/hot carcass weight values reveal that there was more reduction in HCW than there was in rib eye area. For the economic impact, the HCW discount there was significantly less of a discount for "A+O". For QG there was significantly more of a discount for "A+AD" and "A+O". For YG discounts, there was significantly less of a discount for "A", "A+", "A+AD", and "A+O", where only "A-" was a discount and the other significant scores were premiums. For other discounts, sum of premiums and discounts, and market price there were no significant changes for any abnormal liver score. Gross carcass value was significantly lower for all abnormal liver scores. The decrease in gross

carcass value for all even without all having significant discounts and even some premiums, likely is due to the decrease in performance shown in the carcass quality traits (Brown and Lawrence, 2010). While carcass quality and yield decrease, there is no significant impact to meat tenderness and sensory attributes based on liver abscess score (McCoy et al., 2017).

Grimes (2022) had a similar study to Brown and Lawrence (2010) analyzing the effects of liver abscesses on carcass traits in beef cattle. Traits of HCW, back fat thickness, rib eye area, KPH, YG and marbling score. Animals with liver abscesses of all scores were significantly lower than those with non-abscessed livers for HCW, back fat thickness, and rib eye area, with additional decreases as score severity increased. There were no significant differences found in KPH for any liver abscess score compared to one another. For YG, only animals with scores of "A+AD" were significantly lower than YG of any animals with any other score. Marbling score had significant increase between animals with scores of "A+O" and non-abscessed livers, but also additional significant differences between different scores (Grimes, 2022).

Not only do liver abscesses have an impact on carcass performance, but they have also been shown to affect feed efficiency and average daily gain (ADG). Like the effects on carcass traits, the relationship was theorized, but Brink et al. (1990) was the first study to explicitly look at the effects of liver abscess on feed efficiency. Similarly to Montgomery (1985), this study included scores of "A+", "A+AD", and "A+O" into one severe abscess score. The study design included twelve experiments that intended to look at the effects of source and level of crude protein, protein level in connection to rapid change to grain diet, levels of soybean meal, feed additive at different levels, monensin sodium, hay level, corn particle size, corn steep liquor as a lactic acid source, level of defluorinated tricalcium phosphate in connection with grain type, limestone source and calcium level in the diet, and corn silage at 5% in the diet. The twelve

experiments were grouped by variance homogeneity; Group 1 included experiments 3, 7, 8, and 9, whereas Group 2 included experiments 1, 4, 10, 11, and 12. Group 1 had a lower liver abscess incidence than Group 2; rates were 32.1% and 77.7%, respectively. However, there were no clear treatment differences to explain the differences in rates. There were no significant effects on any of the performance traits measured from Group 1. The authors do not hypothesize reasons for Group 1 having no significant differences from abnormal liver abscess scores. Final weight, adjusted final weight, HCW, dressing percentage, daily dry matter intake, daily gain, daily gain calculated with adjusted final weight, and gain/dry matter calculated with adjusted final weight were all significant decreases for scores of "A+" compared to normal livers. Gain/dry matter was the only trait measured that did not show significance for any abnormal liver abscess scores. None of the other abnormal liver abscess scores – "A-" or "A" – had significant effects on the traits (Brink et al., 1990). A notable portion of the experimental design is that for final weight and daily gain, they were tendencies rather than significance, with p-values of above 0.05. Overall, the study solidified that severe liver abscesses do have a relationship to a decrease in feed efficiency and reduced carcass yield.

Feed efficiency was also evaluated in a study by Fox et al. (2009) between two types of vaccines for liver abscesses, but in doing so, also studied feed efficiency for abnormal liver abscess scores. The animals were either given no liver abscess related vaccine (control), given a vaccine containing a *Fusobacterium necrophorum* bacterin, or given a vaccine containing a *Trueperella pyogenes-Fusobacterium necrophorum* toxoid. While the vaccine treatment was not significant in impacting liver abscess scores, they isolated the effects of the liver abscesses on certain feed efficiency and carcass traits. This study also does not include the categorization of A+AD or A+O. There was no significant difference for any liver abscess score for days on feed,

60-day ADG, initial body weight, or 60-day body weight (Fox et al., 2009). This paper states that it is a tendency, but still an important note when comparing the two studies. A statistical decrease was found for A+ for HCW, which is consistent with the literature (Montgomery, 1985; Brown and Lawrence, 2010; Brink et al., 1990). This study looked at USDA quality grade as well, indicating that there was a decrease in the proportion of USDA Choice grades compared to USDA Select (Fox et al., 2009).

## SECTION 7: DIAGNOSIS OF LIVER ABSCESSES

The diagnosis of liver abscesses has proven to be difficult. When cattle have liver abscesses, they typically do not show clinical signs. Some may experience decreased performance, feed efficiency, anorexia, or diarrhea when they have a liver abscess, but this is also commonly associated with the precursor to liver abscess formation – ruminal acidosis (Doré et al., 2007). The connection to ruminal acidosis may overlook the diagnosis potential of liver abscesses. Sudden death may precede diagnosis and will not assist the producer in diagnosing the liver abscesses and treatment prior to slaughter (Doré et al., 2007; Rubarth, 1960). The lack of clinical signs prevents producers from identifying and diagnosing the issue, with most common diagnosis of liver abscess being at necropsy or viscera inspection following naturally occurring death, euthanasia, or slaughter (Macdonald et al., 2017). Diagnosis tools that may be used prior to slaughter are blood testing or ultrasound (Macdonald et al., 2017; Braun, 2009).

Blood testing for the diagnosis of liver abscesses in cattle varies in success yet is continuously studied in the attempts to determine what tests should be run to help diagnose and treat liver abscesses earlier (Macdonald et al., 2017; Doré et al., 2007). Due to the impact on the

immune system and the high metabolic function of the liver, studies commonly run tests on immune response and metabolic functions; however, not all studies perform the same blood tests (Macdonald et al., 2017; Doré et al., 2007; Lechtenberg and Nagaraja, 1991). The three primary studies on blood testing for liver abscess diagnosis in cattle are Macdonald et al. (2017), Doré et al. (2007), and Lechtenberg and Nagaraja (1991). The results from Doré et al. (2007) did not find consistent abnormal results for all 18 abscessed animals. All three studies found a decrease in albumin production, which is a product of protein metabolism in the liver. Doré et al. (2007) and Lechtenberg and Nagaraja (1991) found similar results; however, they also had more overlap in the tests run. Both studies found an increase in neutrophils and leukocytes, fibrinogen, serum globulin concentrations, serum total bilirubin concentrations, and gamma-glutamyltransferase. Lechtenberg and Nagaraja (1991) also found an increase in sorbitol dehydrogenase concentrations and a decrease in sulfobromophthalein clearance. Macdonald et al. (2017) and Doré et al. (2007) had inconsistencies with some tests. While Macdonald et al. (2017) found an increase in aspartate aminotransferase during the 56-day sampling period and a decrease in alkaline phosphatase at slaughter. Doré et al. (2007) found no abnormal results for either test. Macdonald et al. (2017) also found varying results between their samples taken over the span of 56 days and those taken at slaughter. However, the only consistent results between the two were a decrease in albumin and a decrease in cholesterol. While the consistent decrease in albumin across all studies may be beneficial for a blood test to diagnose liver abscesses, a decrease in albumin may also indicate other hepatic diseases and might be difficult to state a clear diagnosis of liver abscesses (Macdonald et al., 2017). While blood tests may be useful in the diagnosis of liver abscesses, the testing methods are not always consistent and do not provide explicit diagnosis of abscesses.

Ultrasound technology can be utilized to help with diagnosis. However, there can be complications with its application. The visualization of liver abscesses is possible and there is the ability to see the progression of the disease using an ultrasound (Braun, 2009). Complications arise in full visualization of the liver in cattle due to the cranial aspect of the liver being covered by the lung – potentially 20% of liver volume can be covered by the lung –, the liver loses contact from the wall of the abdomen when ruminal fill is low, and excess fat may interfere with liver visualization (Braun, 2009; Nagaraja and Lechtenberg, 2007). In addition to issues in visualization, in a production setting, ultrasonography of animals – especially when lacking clinical signs – may be economically inefficient and unreasonable (Nagaraja and Lechtenberg, 2007). However, when ultrasound is feasible, it can be useful. Braun (2009) provides a detailed description of the visualization of liver abscesses when in different stages. In the visualization of liver abscesses, the echogenicity of the content varies; some have no echogenicity, and some have high echogenicity. In early stages of liver abscess formation, there is a heterogeneous ultrasound where there is no capsule and there are one or more abscesses with high echogenicity. In the later stages of liver abscess formation, the ultrasound is homogenous, there is a capsule, and the abscesses are larger. Abscesses may begin separately but can fuse together to form one larger abscess. Partial destruction of the liver has occurred if the abscess itself contains chambers (Braun, 2009).

## SECTION 8: LIVER ABSCESS TREATMENT

Due to the difficulty of diagnosing liver abscesses premortem, the focus on treatment options is limited. However, case studies have evaluated the efficiency of antibiotics and

drainage as treatment options. For antibiotics, the use of a broad-spectrum antibiotic – such as amoxicillin, penicillin, and ampicillin – can effectively treat liver abscesses over the duration of 14 to 23 days (Doré et al., 2007; Braun, 2009). The use of antibiotics in the Doré et al. (2007) study was found to be successful in dairy cows, and following treatment, 71% had adequate milk production, and 57% had a calf and stayed in the herd for an additional one to five years. Drainage of the abscess is possible, primarily if there are singular large abscesses close to the abdominal wall. It can be done using an ultrasound to guide the procedure (Braun, 2009). Doré et al. (2007) attempted drainage on two cattle; however, only one was successful because the other one ruptured. If the abscess is further from the abdominal wall, a laparotomy may be successful in accessing the abscess (Braun, 2009; Fubini et al., 1985). Drainage may not be ideal for cattle with numerous abscesses on the liver and does not treat abscesses present on other organs (Braun, 2009). While treatment using antibiotics is the most effective, the difficulty of premortem diagnosis can prevent timely treatment. Liver abscesses can heal without treatment within 46 and 183 days (Jensen et al., 1954b).

## SECTION 9: LIVER ABSCESS PREVENTION

The current methods of liver abscess prevention in cattle rely on antimicrobial feed additives and acidosis prevention through sound nutrition and vaccination. However, studies have begun to focus on alternatives and introduce a genetic component to liver abscess susceptibility.

The most common prevention technique in conventional feedlots is antimicrobial feed additives. The prophylactic antimicrobial feed additives approved for use in treating liver abscess

in cattle in the United States are tylosin, oxytetracycline, the combination of neomycin and oxytetracycline, chlortetracycline, virginiamycin, and bacitracin (Theurer and Amachawadi, 2022; FDA, 2023a). Tylosin, oxytetracycline, neomycin and oxytetracycline, chlortetracycline, and virginiamycin require a Veterinary Feed Directive to be used in livestock. Bacitracin does not require a Veterinary Feed Directive. (FDA, 2023b). Tylosin is the most common and most efficient antimicrobial feed additive. Bacitracin is the most inefficient antimicrobial feed additive (Tadepalli et al., 2009).

Tylosin is not only the most efficient antimicrobial feed additive, but it is also the most common. Tylosin is a macrolide antibiotic that primarily affects gram-positive bacteria (Papich, 2016). While *T. pyogenes* is susceptible to tylosin due to its categorization as a gram-positive bacterium, *F. necrophorum* is also susceptible to tylosin even though it is a gram-negative bacterium (Nagaraja and Chengappa, 1998). In animals on high concentrate diets, animals receiving tylosin had significantly lower levels of *F. necrophorum* – 80% to 90% less – than those receiving no antimicrobial feed additives. The fermentation products were not significantly affected by the tylosin (Nagaraja et al., 1999). Feeding of tylosin not only decreases *F. necrophorum* concentrations, but also decreases liver abscess incidence. A meta-analysis of six studies regarding tylosin use revealed a significant decrease between those not receiving tylosin and those that are, with respective incidence rates of 30% and 8% (Wileman et al., 2009). The reduction seen by tylosin on liver abscesses is not an elimination of liver abscesses.

A meta-analysis of 17 studies reveals consistent results of a significant decrease in liver abscess prevalence but also reveals a significant decrease in A+ liver abscess scores, meaning tylosin has a significant impact on liver abscess severity (Theurer and Amachawadi, 2022). While tylosin has been proven to have a significant decrease in liver abscess prevalence,

monensin – an ionophore – has been studied in combination with tylosin. The combination has shown to be effective in decreasing liver abscesses, yet monensin fed by itself is not effective in decreasing liver abscesses (Meyer et al., 2013). Tylosin has also proven beneficial to producers based on the increase in the performance of cattle. Brown et al. (1975) reveals a significant improvement in average feed conversion for animals fed tylosin compared to the control, with feed conversions of 7.87 and 8.21, respectively. Average daily gain was not significantly higher for tylosin compared to the control, yet numerically there was a slight increase (Brown et al., 1975). Vogel and Laudert (1994) found a significant increase in ADG and dressing percentage for those fed tylosin, and a significant decrease in feed to gain ratio, which indicates improvement. Not all studies report significant performance improvement in cattle on tylosin. Müller et al. (2018) report no significant difference between animals on continuous or intermittent tylosin for any feed efficiency or carcass quality or yield traits except for marbling score, which decreases significantly on continuous tylosin.

The other antimicrobial feed additives vary in efficiency and in amount studied. Oxytetracycline was studied by Lee and Laudert (1984) and found no significant effect on liver abscess prevalence. The numerical trend revealed an increase in liver abscess incidence rate from the control to those treated with oxytetracycline of 19% to 25%, respectively (Lee and Laudert, 1984). Neomycin and oxytetracycline have not been studied; however, it is approved by the Food and Drug Administration (FDA) for use in cattle with the intention of decreasing liver abscesses (Flynn, 2009; Theurer and Amachawadi, 2022). Chlortetracycline has been shown to have a significant decrease in liver abscess prevalence. Brown et al. (1975) and Wieser et al. (1966) reveal significant decreases to liver abscess prevalence and improvement of feed efficiency and ADG. Brown et al. (1975) studied both tylosin and chlortetracycline, so while both did show a

significant decrease in liver abscess prevalence from the control, chlortetracycline had a 12-percentage point decrease, and tylosin had a 37.6-percentage point decrease. Virginiamycin is the second most studied antimicrobial feed additive, yet it has lower commercial use due to its inability to be used in combination with other feed additives (Theurer and Amachawadi, 2022). Tedeschi and Gorocica-Buenfil (2018) found that cattle fed virginiamycin had a significant decrease in liver abscess prevalence, from 19.3% in the control to 11.2% in those treated and in liver abscess severity. Latack et al. (2019) did not find a significant difference between the control and those treated with virginiamycin, yet numerically there was a decrease. Bacitracin is the most ineffective, having no effect on liver abscess incidence rate, with both groups having a 72% incidence rate (Haskins et al., 1967). While there are six antimicrobial feed additives approved for use in the United States to prevent liver abscesses, not all are effective or heavily utilized commercially.

While antimicrobial feed additives are popular amongst conventional producers, and some have been proven to be efficient, current production systems may have to turn to alternatives due to increased concerns about antimicrobial resistance, verification services, and consumer perception. Agga et al. (2023) and Cazer et al. (2020) found increases in macrolide resistant enterococci in animals on continuous feeding of tylosin. Macrolide resistance is important to the industry due to the bacteria not being susceptible to the antibiotics, but also because it has the potential to contaminate beef and impact humans. Agga et al. (2023) specifically found the presence of *Enterococcus faecium* and *Enterococcus faecalis*, the most important pathogenic agents when considering human infection. The United States Department of Agriculture (USDA) has been taking steps to address antimicrobial resistance with the focus of One Health – an approach including people, animals, plants, and the environment – in mind

(USDA, 2023). Decreasing the use of antibiotics is a strategy in this plan. Verification services regarding antibiotic use have also grown in popularity. USDA Organic is a verification service that has requirements that need to be met by producers and audited to grant verification. The USDA Organic verification has numerous requirements, but one is that animals must be managed without antibiotics or other prohibited feed ingredients (USDA, 2013). This requirement means that producers cannot use antimicrobial feed additives as a method of prevention of liver abscesses and need to explore alternate avenues. While conventional producers do not face the requirements that organic producers face, the move away from antimicrobial feed additives may eventually affect them due to consumer preferences. Consumer demand plays a large role in the supply chain. A meta-analysis reveals that 77.1% of consumers ranging from studies in the United States, Canada, and the European Union have concerns over antimicrobial use in the production of meat, with human health and animal welfare being specifically stated concerns (Barrett et al., 2021). As time continues, consumers seem to have increased concerns over antimicrobial use and that may impact the use of antimicrobial feed additives.

Nutrition management to prevent liver abscess incidence is a significant prevention technique due to the establishment of the rumenitis-liver abscess complex. The decrease in acidosis occurrence leads to a decrease in rumenitis and, therefore, a decrease in liver abscess development. Owens et al. (1998) outline the danger scale for acidosis development of different nutritional management, diet composition, and feed additives. Low acidosis risk is present when cattle are fed small meals with limited feed access, have low dietary concentrate levels, less readily fermentable carbohydrates, unfermented feed, high protein, up to 8% fat, and an acidic dietary cation-anion balance. Feed additives such as ionophores, bicarbonate, probiotics and probiotic stimulants, thiamin, and antimicrobials also assist in decreasing acidosis risk (Owens et

al., 1998). In addition, increased roughage in the diet may help decrease acidosis risk (Huntington, 1988). Maintenance of a diet that has a low acidosis risk proves difficult in feedlot and dairy settings. The increased energy provided from high concentrate diets benefits feedlot and dairy producers due to the increased gain seen in the animals. With the proper diet formulation, meal size, and introduction to high concentrate diets, acidosis prevalence in cattle can be decreased (González et al., 2012).

Vaccinations as a preventative measure for liver abscesses in cattle have been studied since 1954, when Jensen et al. (1954b) looked at immunization using *F. necrophorum* following experimental inoculation. Vaccination against the experimental inoculation was not successful, with no significant difference detected between those given the vaccination and the control (Jensen et al., 1954b). Checkley et al. (2005) found inconsistent success of vaccinations, revealing a vaccine and diet interaction when given the *F. necrophorum* vaccination. The animals on a limit fed grain diet had no significant difference in liver abscess incidence, with incidence rates of 7.1% and 7.4% for vaccinated and unvaccinated animals, respectively. Conversely, the animals on an ad libitum forage diet had a significant decrease in liver abscess incidence, with incidence rates of 2.6% and 9.3% for vaccinated and unvaccinated animals, respectively (Checkley et al., 2005). The vaccine and diet interaction indicates that the nutritional management of the animals may play a larger role, especially seen in the limit fed grain diet's acidotic effect.

Subsequent studies on vaccinations reveal similar results, with Fox et al. (2009) finding no significant difference for liver abscess incidence rate or severity in cattle given a vaccination of *F. necrophorum.* Fox et al. (2009) tested the efficiency of a *F. necrophorum* leukotoxin and *T. pyogenes* bacterin vaccination, also finding no significant differences in liver abscess incident

rate or severity. The *T. pyogenes – F. necrophorum* bacterin-toxoid vaccination was first studied on a large basis by Jones et al. (2004). The literature included two studies; the first study included animals immunized that were not on medicated feed and the second study included immunized animals either on or off medicated feed. The first study found that animals given a high antigen dose of the vaccination had significantly lower liver abscess incidence rate, however, a low antigen dose of the vaccination had no significant difference. The second study found that both high antigen dose and low antigen dose vaccination were significantly lower in liver abscess incidence for animals on non-medicated feed. The animals on medicated feed in the second study, given the low antigen dose vaccination, had significantly lower liver abscess incidence (Jones et al., 2004). Overall, studies on vaccination efficiency were inconsistent.

One vaccination is commercially available for the prevention of liver abscesses. Fusogard is a *F. necrophorum* vaccination produced by Elanco (Elanco Animal Health, Greenfield, IN). Fusogard is also directed for use in the prevention of foot rot due to the common pathogenic agent. The *T. pyogenes – F. necrophorum* bacterin-toxoid vaccination was previously available for commercial use, produced by Merck Animal Health (Merck Animal Health, Rahway, NJ), however, it was discontinued. More studies need to be conducted in the search for a consistent and efficient vaccination for liver abscesses, however, vaccination utilization may be a beneficial form of prevention due to its ability for use in all production systems (Amachawadi and Nagaraja, 2016).

Accounting for the increase in negative public perception of antimicrobial feed additives, the potential difficulty in nutritional consistency, and the low efficiency of vaccinations in preventing liver abscesses, a study by Keele et al. (2016) performed a genome-wide association study, initiating investigations into a potential genetic pathway for prevention. Keele et al. (2016)

performed their study by analyzing the DNA of beef cattle, half of the samples taken from animals with liver abscesses and half taken from animals with normal livers. The study found 35 SNP to be associated with liver abscesses. The SNP were on 17 autosomal chromosomes and the X chromosome. The SNP that were detected and polygenic variation without effect on phenotypic variation found in the study led to the determination that there is partial genetic control of liver abscesses. Keele et al. (2016) mentions the current trend in the selection for heavier and faster-gaining cattle contributing to increased liver abscess incidence. Heaver animals typically eat more and are more susceptible to acidosis and, ultimately, liver abscesses. The genes near the SNP had different functions contributing to liver abscess formation and susceptibility. Pathways included pH homeostasis in the gastrointestinal tract, maintenance of liver immunity, leukocyte transport into infected tissue, production of bicarbonate during acidosis through glutamine transport into the kidney, axon guidance, and assistance of liver repair through aggregation of platelets (Keele et al., 2016). The finding of partial genetic control indicates that there may be a potential for liver abscesses to be heritable and subject to genetic selection. The ability for genetic selection utilization may provide a new prevention technique that can be implemented prior to conception, reduce needs for antimicrobials, and reduce overall liver abscess incidence.

## SECTION 10: GENETIC EVALUATION OF DISEASE TRAITS

Genetic evaluation and subsequent selection of many non-genetic disease traits is possible and has been done for numerous cattle diseases. For genetic defect diseases such as arthrogryposis multiplex and congenital contractural arachnodactyly in Angus, the associated

genes have been identified, allowing for prevention through genetic testing (Whitlock et al., 2008). While susceptibility to multifactorial and polygenic diseases is not as directly identifiable as monogenic diseases, there is an ability to identify a heritability for susceptibility to the disease. In the dairy industry, health traits such as displaced abomasum, ketosis, mastitis, lameness, cystic ovaries, metritis, pneumonia, and many more have been identified as low to moderately heritable (Lyons et al., 1991; Zwald et al., 2004). Even health categories, such as reproductive, mammary, digestive, etc. have been reported to have heritability estimates ranging from 0.02 to 0.21 (Lyons et al., 1991). In beef cattle, bovine respiratory disease has also been found to be lowly heritable with estimates ranging from 0.04 to 0.2 (Hayes et al., 2024; Cockrum et al., 2016; Schneider et al., 2010; Snowder et al., 2005; Muggli-Cockett et al., 1992). Having non-zero heritability estimates for these disease traits does allow producers to be able to utilize genetic selection to assist in the decrease of the disease incidence in their herd. Through the use of genetic evaluation and data collection, the genetic selection of animals and bloodlines with less incidence of disease may be used to produce more resilient replacements.

While heritability estimates have been published for numerous polygenic and multifactorial diseases, there are underlying limitations that can make genetic evaluations for disease traits complicated. Berry et al. (2011) outlines the limitations commonly seen when evaluating health traits. Difficulties revolving around data collection include error in diagnostics and phenotyping, sex-limited diseases, and diseases that appear later in life. Statistical difficulties include genotype by environment interactions and need for more information to estimate with higher accuracy. There may also be antagonistic genetic correlations that complicate selection decisions for a producer (Berry et al., 2011). Additionally, the inability to know exactly when an animal was exposed to a pathogen and to how much produces difficulty in measurement of that

disease response. Limitations most generically relevant to liver abscess genetic evaluation in cattle would be the current lack of antemortem diagnostics. Animals are typically only phenotyped at slaughter, which increases the length of time until the disease is diagnosed and repeated records would be unlikely in current producer data. Knowledge of successes and limitations in genetic evaluations of disease traits in cattle can help contribute to the proper development of a genetic selection tool for liver abscesses in cattle.

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# CHAPTER 3: THE IMPACT OF LIVER ABSCESS SEVERITY ON CARCASS TRAITS IN BEEF ON DAIRY HEIFERS

## SUMMARY

The economic impacts of liver abscesses on the beef industry due to liver and gut condemnation are well reported. However, inconsistent reports of decreases in animal performance and carcass quality both between and within studies exist. The objective of this study was to determine the effects of liver abscess severity on hot carcass weight (HCW; kg), rib eye area (REA; $cm^2$), back fat thickness (FT; cm), marbling score (MS), calculated visual yield grade (VYG), and heart score (HS) in a population of fed beef on dairy heifers. All 1,860 heifers used in this study were fed and harvested in Kansas. At harvest, phenotypes for HCW, REA, FT, MS, VYG, and liver abscess scores were collected on all heifers. Phenotypes for HS were collected on only 1,646 of the heifers. Univariate multiple linear regression models were used to evaluate the phenotypic relationship between carcass traits (HCW, REA, FT, MS, VYG, and HS) and liver abscess traits. Models for HCW, REA, and MS included fixed effects of liver abscess score, contemporary group, age in days, and feedlot entrance weight in kg. Models for FT and VYG included fixed effects of liver abscess score, contemporary group, age in days, feedlot entrance weight in kg, and number of bovine respiratory disease treatments. The model for HS fit fixed effects of liver abscess score, contemporary group, and age in days. Contemporary group was a concatenation of kill lot and treatment. Liver abscess phenotypes were assigned using two approaches: one with six unique liver abscess scores and one with four unique liver abscess scores that condensed scores of “A+”, “A+AD”, and “A+O” into the “A+” category. A significant increase in FT was identified using the six-score model for animals with scores of

"A+O" having higher FT compared to "A+", with respective least-squares means of 1.94 cm ± 0.12 and 1.59 cm ± 0.06 ($P < 0.05$). There were non-significant differences that were tendencies for "A" and "A+AD" compared to "A+O" (1.61 cm ± 0.06, 1.61 cm ± 0.05, and 1.94 cm ± 0.12, respectively; $0.05 \leq P < 0.10$). A significant increase in VYG was observed using the six-score model for VYG with scores of "A+O" compared to "A+" and "A+AD" (3.75 ± 0.19, 3.20 ± 0.09, and 3.20 ± 0.08, respectively; $P < 0.05$). The models with the four-score system revealed a significant decrease in HCW for animals with scores of "A+" versus scores of "0", with least-squares means of 391 kg ± 2.92 and 396 kg ± 2.63, respectively ($P < 0.05$). These results reveal that liver abscesses have a significant effect on HCW with more severe levels reducing HCW.

## INTRODUCTION

The economic impact of liver abscesses on the beef industry can be directly tied to the condemnation of livers and gut contents. Liver abscess incidences for fed cattle of dairy or beef backgrounds range from 12% to 48% (Brown and Lawrence, 2010; Amachawadi and Nagaraja, 2016; Eastwood et al., 2017; Herrick et al., 2022). In 2009, the annual estimated loss in the United States due to liver abscesses was $15.8 million, with an incidence rate of 18.1% in fed cattle. In the same study, an additional $7.0 million was lost annually due to animals with liver adhesion and abscess rupture, with an incidence rate of 3.5% (Brown and Lawrence, 2010). A separate study found the estimated economic loss from 2015 to 2016 was $46.1 million due to liver abscesses (Herrick et al., 2022). Economic factors due to condemnation are the most direct; however, decreases in carcass quality and performance have also contributed to additional

discounts. These decreases can be due to the carcass quality, but also a decrease in processing efficiency if there is a need to do extra trimming or condemnation.

Numerous studies have investigated the impacts of liver abscesses on final product weight, yield, and quality. Decreases in carcass weight, yield, and quality grade can reduce overall income through loss of premiums or application of discounts. Significant findings across studies identify undesirable impacts on HCW, REA, MS, and quality grade with increased abscess severity (Montgomery, 1985; Brink et al., 1990; Fox et al., 2009; Brown and Lawrence, 2010; Rezac et al., 2014), but these are not consistent and therefore more research to determine what impacts liver abscesses consistently have on carcass traits is needed.

The objective of this study was to provide an analysis of the effects of liver abscess severity on hot carcass weight (HCW; kg), rib eye area (REA; $cm^2$), back fat thickness (FT; cm), marbling score (MS), calculated visual yield grade (VYG), and heart score (HS) in a population of fed beef on dairy heifers.

## MATERIALS AND METHODS

The data was collected from 1,860 beef on dairy heifers fed and harvested in Kansas. Harvest dates were from 2022 and 2023. At the feedlot, the heifers were split into separate lots, and each lot was assigned to one of three treatments. The treatments were part of a larger study analyzing the efficacy of different feed additives in liver abscess mitigation. Since our study did not have the same objective, the treatment was not of direct statistical interest. To account for the variation attributed to these treatments, the treatment was combined with lot to form contemporary group and included in all models. Records of feedlot in-weight in kg and number

of bovine respiratory disease (BRD) treatments were collected at the feedlot. At harvest, phenotypes for hot carcass weight (HCW; kg), rib eye area (REA; $cm^2$), back fat thickness (FT; cm), marbling score (MS), calculated visual yield grade (VYG), heart score (HS), and liver abscess score were collected. All 1,860 individuals had phenotypes recorded, but of these, only 1,646 animals had observations for HS. Heart score was scored by a trained evaluator using the system developed by Dr. Tim Holt at Colorado State University. The range is from 1 to 5, with the severity of heart remodeling increasing as the numerical score increases. Hearts with a score of 1 are physiologically normal and those with a score of 5 have severe remodeling, specifically in the right ventricle (Heffernan et al., 2020; Kukor et al., 2021). A trained evaluator scored liver abscesses using the Elanco Liver Scoring System (Elanco Animal Health, Greenfield, IN) as described in Table 3.1.

Table 3.1 Liver abscess scores and descriptions based on the Elanco Liver Scoring System (Elanco Animal Health, Greenfield, IN).

| Liver Abscess Score | Description |
|---|---|
| 0 | No abscess present. |
| A- | One or two abscesses that are less than 2.5 cm in diameter. |
| A | Numerous abscesses that are less than 2.5 cm in diameter OR one or two abscesses greater than 2.5 cm in diameter. |
| A+ | Numerous abscesses greater than 2.5 cm in diameter. |
| A+AD | Liver adhered to a nearby organ. No specific abscess size or amount. |
| A+O | Liver abscess ruptured. No specific abscess size or amount. |

Model development and analyses were conducted utilizing the statistical software package R (R Core Team, 2023), RStudio (Posit team, 2024), and the *emmeans* package (Lenth R, 2024). Two univariate, multiple linear regression models were fit for each carcass trait of interest as the dependent variable (HCW, REA, FT, MS, VYG, and HS). Each of the models was run twice to include different variations of the liver abscess scoring system. The first set of models included all six unique liver abscess scores, referred to as the "six-score system" ("0",

"A-", "A", "A+", "A+AD", and "A+O"). In the second set of models, scores of "A+", "A+AD", and "A+O" were consolidated into "A+", so there were only four unique liver abscess scores, referred to as the "four-score system" ("0", "A-", "A", and "A+"). The combination of A+, A+AD, and A+O was done to analyze the more severe categories as one to better compare to previous studies that have "A+" including those with adhesion and rupture. To determine the models, full models that included fixed effects of liver abscess score, contemporary group, age in days, feedlot in-weight, and number of BRD treatments were fit to each carcass trait. Overall variable significance in the model was tested using a Type III ANOVA, where $P < 0.05$. Tendencies were identified when $0.05 \leq P < 0.10$. Regardless of the significance of the model, all models included liver abscess, contemporary group, and age. Liver abscess score was always included due to the objective of evaluating the relationship between the carcass traits and liver abscess score. Contemporary group and age were always included to account for known environmental effects. Contemporary group was a combination of treatment and lot.

Least-squares means were estimated for each trait of interest at all liver abscess score levels, adjusting for the average contemporary group, age in days, and any additional fixed effects depending on the model used. Pairwise contrasts were conducted to evaluate for differences across all liver abscess score levels when the overall significance of liver score in the model was $P < 0.05$. Significance was determined using Tukey-adjusted P-values provided from the pairwise contrasts. Tendencies were identified when $0.05 \leq P < 0.10$. Significance was determined at $\alpha = 0.05$.

# RESULTS AND DISCUSSION

The minimum, maximum, mean, and variance for all carcass traits are shown in Table 3.2. The averages and ranges for HCW (kg), REA ($cm^2$), FT (cm), and MS lie within those reported for fed beef on dairy animals (Keele et al., 2024). The VYG in this data set has a higher maximum than that reported by Keele et al. (2024) of 5.60 for beef on dairy. The lack of male animals in this data set may contribute to numerical differences. As of the time of writing, beef on dairy heart score statistics have not been published. Kukor et al. (2021) reported an average heart score for beef cattle of 2.30, which was higher than seen in this data set. One animal had a heart score of 5 in this study, indicating severe heart failure.

Table 3.2 Number of observations (N), mean, standard deviation, minimum, and maximum values for carcass characteristics from the beef on dairy heifers in the study.

| | N | Mean | Standard Deviation | Minimum | Maximum |
|---|---|---|---|---|---|
| Hot Carcass Weight, kg | 1860 | 387.11 | 38.03 | 220.45 | 521.18 |
| Rib Eye Area, $cm^2$ | 1860 | 93.36 | 9.47 | 62.58 | 124.52 |
| Back Fat Thickness, cm | 1860 | 1.66 | 0.48 | 0.30 | 5.49 |
| Marbling [1] | 1860 | 524.92 | 104.01 | 332 | 930 |
| Visual Yield Grade | 1860 | 3.25 | 0.77 | 1 | 6.86 |
| Heart Score | 1646 | 1.72 | 0.68 | 1 | 5 |
| Age, days | 1860 | 519.06 | 36.67 | 404 | 676 |

[1]Leading digit in marbling indicates score; 2=trace, 3=slight, 4=small, 5=modest, 6=moderate, 7=slightly abundant, 8=moderately abundant, 9=abundant. The following digits indicate the degree of marbling within a marbling score

For the development of the models utilized for each carcass trait, overall significance of variables was tested. Table 3.3 and 3.4 report the P-values for each potential variable in the model per carcass trait when using the four-score and six-score systems, respectively, from the Type III ANOVA. All models contained liver abscess score, contemporary group, and age in

days. The addition of in-weight and number of BRD treatments were tested, so if $P < 0.05$ then those additional variables were included in the model. Contemporary group was significant in all 12 models ($P < 0.05$). Age and in-weight were significant in all models except the two HS models ($P < 0.05$). Number of BRD treatments was significant in the FT and VYG models ($P < 0.05$). Liver abscess score significance was not always the same on the four and six-score systems. In both systems, liver abscess score was not significant for REA, MS, and HS ($P > 0.05$). In the four-score system, liver abscess score was significant for hot carcass weight ($P < 0.05$), but there was a tendency in the six-score system ($P = 0.06$). Liver abscess score was not significant on the four-score system for FT and VYG ($P > 0.05$) but was significant on the six-score system ($P < 0.05$).

Table 3.3 P-values for fixed effects in overall carcass trait model from Type III ANOVA. Liver abscess score used was the four-score system.

| | Liver Abscess Score (4-score) | Contemporary Group | Age | In-weight | Number of BRD treatments |
|---|---|---|---|---|---|
| Hot Carcass Weight | 0.02 * | < 0.001 * | < 0.001 * | < 0.001 * | 0.39 |
| Rib Eye Area | 0.60 | < 0.001 * | < 0.001 * | < 0.001 * | 0.81 |
| Back Fat Thickness | 0.12 | < 0.001 * | < 0.001 * | < 0.001 * | < 0.001 * |
| Marbling Score | 0.76 | < 0.001 * | < 0.001 * | 0.01 * | 0.71 |
| Visual Yield Grade | 0.15 | < 0.001 * | < 0.01 * | < 0.001 * | 0.03 * |
| Heart Score | 0.11 | 0.005 * | 0.67 | 0.80 | 0.88 |

* Significant with $P < 0.05$.

Table 3.4 P-values for fixed effects in overall carcass trait model from Type III ANOVA. Liver abscess score used was the six-score system.

| | Liver Abscess Score (6-score) | Contemporary Group | Age | In-weight | Number of BRD treatments |
|---|---|---|---|---|---|
| Hot Carcass Weight | 0.06 [1] | < 0.001 * | < 0.001 * | < 0.001 * | 0.41 |
| Rib Eye Area | 0.42 | < 0.001 * | < 0.001 * | < 0.001 * | 0.79 |
| Back Fat Thickness | 0.01 * | < 0.001 * | < 0.001 * | < 0.001 * | < 0.001 * |
| Marbling Score | 0.60 | < 0.001 * | < 0.001 * | 0.01 * | 0.70 |
| Visual Yield Grade | 0.02 * | < 0.001 * | < 0.01 * | < 0.001 * | 0.03 * |
| Heart Score | 0.29 | < 0.01 * | 0.67 | 0.81 | 0.89 |

[1] A tendency with $0.05 \leq P < 0.10$
* Significant with $P < 0.05$

Models used for HCW, REA, and MS had fixed effects of liver abscess score, contemporary group, age, and in-weight. Models used for FT and VYG had fixed effects of liver abscess score, contemporary group, age, in-weight, and number of BRD treatments. Models used for HS had fixed effects of liver abscess score, contemporary group, and age.

The liver abscess incidence was 39.5%, with the distribution of liver abscess scores shown in Figure 3.1. Reported beef on dairy liver abscess incidence remains higher than most fed beef or dairy cattle, with an incidence rate of 50.18% (Grimes, 2022). The rate in this study was not as high, but does remain higher than most reported rates in fed beef cattle and dairy cattle. The incidence rate in this study lies nearer the reported values from Brown and Lawrence (2010) for their database of beef cattle without any fed preventatives of 42.8%. Management styles differed in cattle type as well as preventative use because some animals in our study were on oral preventatives. However, the incidence rate for those from a similar management system with use of fed preventatives to this study was 12.0% (Brown and Lawrence, 2010). Fed beef

cattle incidence rates average around 20% (Amachawadi and Nagaraja, 2016; Herrick et al., 2022). Fed Holstein cattle incidence rates average around 25% (Herrick et al., 2022). The specific cause for higher incidence rates in beef on dairy cattle has not been identified. Dairy cattle also having higher incidence rates, and therefore there may be an underlying genetic component or neonate management effect. Broadway et al. (2024) speculates causes for the breed type differences could include genetics, higher levels of feed intake, greater ruminal volume, the animal's growth potential, lessening of barrier function, and geographical region, but regardless it is likely multifactorial (Broadway et al., 2024).

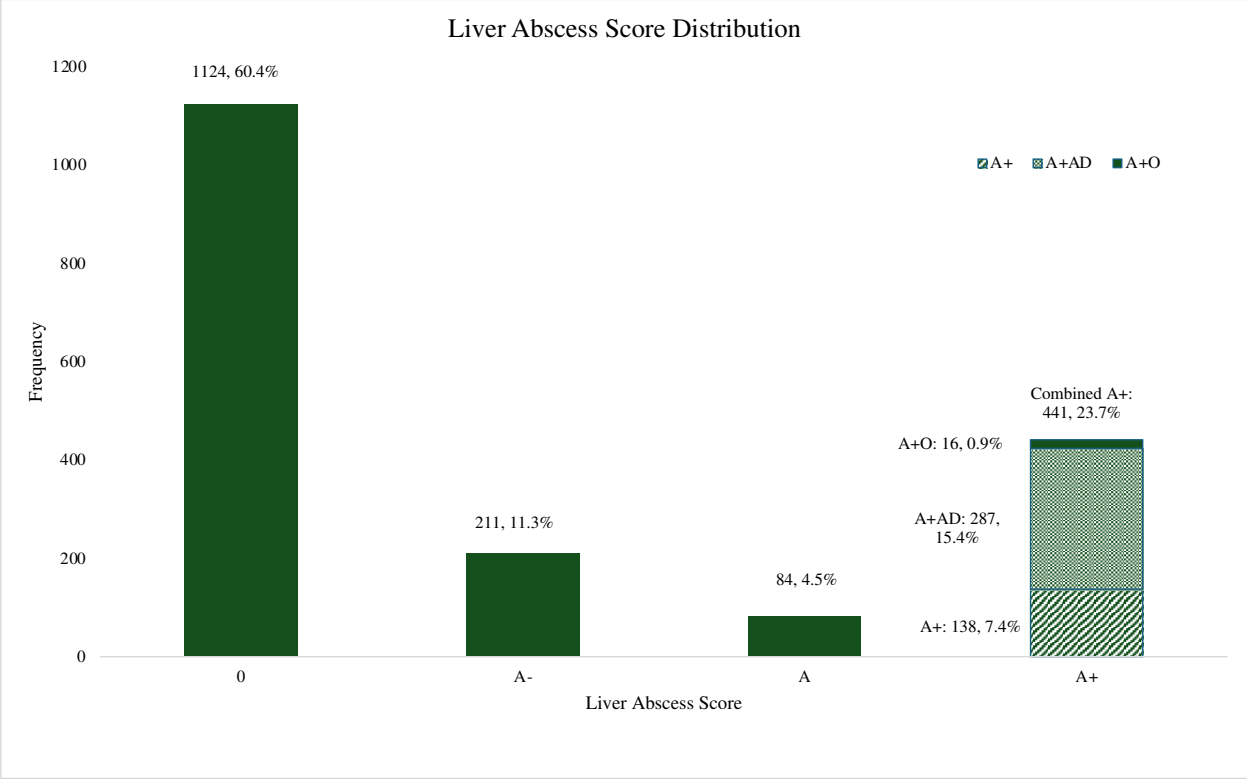


Figure 3.1 Histogram of liver abscess score distributions. Includes distributions for all scores and the combined "A+" score. The percentage of the total was also included.

The least-squares means from the multiple linear regression models with all six liver abscess scores were reported in Table 3.5. There was a significant increase in FT for animals with scores of "A+O" compared to "A+" ($P < 0.05$). The least-squares means for FT for animals with scores of "A+" was 1.59 cm ± 0.06 and "A+O" was 1.94 cm ± 0.12. There were tendencies observed for animals with scores of "A" (1.61 cm ± 0.06) having lesser FT compared to "A+O" (1.94 cm ± 0.12; $P = 0.09$) and animals with scores of "A+AD" (1.61 cm ± 0.05) compared to "A+O" (1.94 cm ± 0.12; $P = 0.07$). A significant increase in VYG was also present between animals with scores of "A+O" compared to "A+AD" and "A+O" compared to "A+" ($P < 0.05$). The least-squares means for VYG for animals with scores of "A+" was 3.20 ± 0.09, "A+AD" was 3.20 ± 0.08, and "A+O" was 3.75 ± 0.19. While liver abscess score did not have a significant effect on HCW, there was a tendency for increasing abscess severity to decrease HCW in the six-score model. Liver abscess score impacts were not tested for significance for the other carcass traits because liver abscess score was not significant in the whole model.

Only one study analyzed data using the six-score system and compared values between scores. In Grimes (2022) FT was identified to also have a significant increase in animals with scores of "A+" and "A+AD" compared to "A+O". However, unlike our study, the animals with "A+O" did not have the highest estimated mean out of all scores. Significant findings in VYG were not as similar. Technically, animals with scores of "A+AD" had significantly lower VYG compared to those with "A+O", but "A+AD" was also significantly lower than all other scores because the abscessed scores had the same estimated means (Grimes, 2022).

Table 3.5 Least-squares means and standard error in parenthesis for the models with carcass traits as the response variable. Overall significance of liver abscess score (expressed as 6 categories) in the model included. All scores are represented individually. Adjusted for contemporary group and age in days.

| | 0 | A- | A | A+ | A+AD | A+O | P-Value |
|---|---|---|---|---|---|---|---|
| N | 1124 | 211 | 84 | 138 | 287 | 16 | |
| Hot Carcass Weight, kg [1] | 396 (2.63) | 392 (3.27) | 392 (4.09) | 389 (3.60) | 391 (3.09) | 397 (7.87) | 0.06 [1] |
| Rib Eye Area, $cm^2$ [1] | 93.8 (0.77) | 93.0 (0.97) | 92.9 (1.21) | 93.2 (1.06) | 94.0 (0.91) | 90.4 (2.32) | 0.41 |
| Back Fat Thickness, cm [2] | 1.67 (0.04) | 1.69 (0.05) | 1.61 (0.06) | 1.59 (0.06) [a] | 1.61 (0.05) | 1.94 (0.12) [a] | 0.01 * |
| Marbling [1,3] | 543 (8.82) | 535 (11.0) | 542 (13.8) | 542 (12.1) | 542 (10.4) | 582 (26.5) | 0.60 |
| Visual Yield Grade [2] | 3.30 (0.06) | 3.33 (0.08) | 3.26 (0.10) | 3.20 (0.09) [a] | 3.20 (0.08) [b] | 3.75 (0.19) [a,b] | 0.02 * |
| Heart Score | 1.70 (0.06) | 1.70 (0.08) | 1.51 (0.10) | 1.71 (0.08) | 1.73 (0.07) | 1.78 (0.20) | 0.29 |

[a-b] Means in the same row that share a common superscript differ ($P < 0.05$)

[1] Additional adjustment for feedlot in weight

[2] Additional adjustment for feedlot in weight and number of bovine respiratory disease treatments

[3] Leading digit in marbling indicates score; 2=trace, 3=slight, 4=small, 5=modest, 6=moderate, 7=slightly abundant, 8=moderately abundant, 9=abundant. The following digits indicate the degree of marbling within a marbling score

* Significant with $P < 0.05$

Brown and Lawrence (2010) also used the six-score system, but compared means to normal livers (i.e. no abscesses). They found significant differences in FT and VYG for abscessed scores compared to normal livers, but we did not find those same differences. In this study, means of FT and VYG of animals with scores of "A+O" had the greatest levels of fatness unlike those in the Brown and Lawrence study.

Increases in FT and VYG are generally undesirable. The yield grade equation includes back fat thickness, so an increase in back fat thickness is associated with increased yield grade. Discounts are commonly applied to higher yield grades of 4 or 5 and animals with high levels of back fat due to the additional trimming required and lower levels of red meat product (Parish et al., 2016; Ward et al., 2017). The cause for FT and VYG increases may be due to consequences of illness or decreased liver function leading to more fat deposition.

The least-squares means from the models with four liver abscess score categories were reported in Table 3.6. A significant decrease in HCW for animals with scores of "A+" compared to non-abscessed animals was identified ($P < 0.05$). The least-squares means for HCW for animals with scores of "0" and "A+" were 396 kg ± 2.63 and 391 kg ± 2.92, respectively. Liver abscess score impacts were not tested for significance for the other carcass traits because liver abscess score was not significant in the whole model. Differences between significance found in the four and six score systems likely relates to the distribution of scores. When "A+" is a grouped score, there are 441 observations; however, when "A+", "A+AD", and "A+O" are individual scores, they have lower respective observations of 138, 287, and 16, respectively.

Table 3.6 Least-squares means and standard error in parenthesis for the models with carcass traits as the response variable. Overall significance of liver abscess score in the model included. Adjusted for contemporary group and age in days.

| | 0 | A- | A | A+ | P-Value |
|---|---|---|---|---|---|
| N | 1124 | 211 | 84 | 441 | |
| Hot Carcass Weight, kg [1] | 396 (2.63) [a] | 392 (3.27) | 392 (4.09) | 391 (2.92) [a] | 0.02 * |
| Rib Eye Area, $cm^2$ [1] | 93.8 (0.77) | 93.0 (0.97) | 92.9 (1.21) | 93.7 (0.86) | 0.59 |
| Fat Thickness, cm [2] | 1.67 (0.04) | 1.69 (0.05) | 1.61 (0.06) | 1.62 (0.05) | 0.12 |
| Marbling [1,3] | 543 (8.84) | 535 (11.0) | 542 (13.8) | 543 (9.83) | 0.76 |
| Visual Yield Grade [2] | 3.30 (0.06) | 3.33 (0.08) | 3.26 (0.10) | 3.22 (0.07) | 0.15 |
| Heart Score | 1.70 (0.06) | 1.70 (0.08) | 1.51 (0.10) | 1.72 (0.07) | 0.11 |

[a] Means in the same row with the same superscripts are significantly different ($P < 0.05$)

[1] Additional adjustment for animal feedlot in weight

[2] Additional adjustment for animal feedlot in weight and number of bovine respiratory disease treatments

[3] Leading digit in marbling indicates score; 2=trace, 3=slight, 4=small, 5=modest, 6=moderate, 7=slightly abundant, 8=moderately abundant, 9=abundant. The following digits indicate the degree of marbling within a marbling score

* Significant with $P < 0.05$

Of the studies that have been conducted using the same four-score system with a combined severe abscess group, there have been similar results reported for HCW in this study. Significant decreases have been reported for scores of "A+" in some studies (Brink et al., 1990; Montgomery, 1985; Fox et al., 2009; Rezac et al., 2014). Rezac et al. (2014) and Fox et al. (2009) found similar results with animals having a score of "A+" having significantly lower HCW when compared to non-abscessed livers. Fox et al. (2009) also identified a significantly lower HCW with animals having a score of "A". Brink et al. (1990) identified a significant decrease in HCW for those with scores of "A+" in only one of the two experimental groupings in that study where groups were based on the diet they were fed. The "A+" category did include

scores of “A+AD” and “A+O”, like this study (Brink et al., 1990). Montgomery (1985) found no significant difference in HCW for any abscessed score in fed Holstein cattle; yet, they found a significant decrease in HCW for fed non-Holstein cattle with scores of “A+” compared to normal and less severe scores. The “A+” category did include scores of “A+AD” and “A+O” as in this study (Montgomery, 1985). A decrease in HCW potentially is due to the animal having an active immune response to the liver abscesses and decreased liver function, and therefore could either have reduced food intake and be partitioning energy towards the immune response over muscle and fat synthesis.

Carcasses with liver abscesses may be underperforming compared to level and cost of input, such as feed, and the actual profit may not meet the expected (Parish et al., 2016). The loss in expected profit is only then exacerbated by the loss due to condemnation of the liver.

## CONCLUSION

Results in this study were not consistent between analysis using a four-score system and a six-score system for liver abscess scores. In the four-score system, HCW was significantly lower in animals with scores of “A+” compared to normal livers, where “A+” was a combination of severe liver abscess scores of “A+”, “A+AD”, and “A+O”. In the six-score system, FT and VYG were significantly higher in animals with scores of “A+O” compared to “A+”, and additionally with scores of “A+O” compared to “A+AD” for VYG. There were also non-significant tendency differences between scores of “A” and “A+AD” with lower VYG when compared to “A+O”. Numerous studies have produced similar results for HCW, however, only one study has produced a similar result for FT. While some similar results for significance and lack of significance have

been found in beef and dairy cattle, there were no studies with a direct and explicit comparison for beef on dairy cattle. The lack of beef on dairy literature and the inconsistencies of beef cattle literature necessitates further research on the impacts of liver abscess severity on different fed cattle types. However, implications of negative performance impacts on HCW, FT, and VYG in animals with severe abscesses indicate an economic concern.

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# CHAPTER 4: HERITABILITY ESTIMATES FOR LIVER ABSCESS INCIDENCE AND SEVERITY IN FED BEEF ON DAIRY HEIFERS

## SUMMARY

Due to the lack of outward clinical signs of liver abscesses in fed cattle, diagnosis remains costly and not feasible in production settings. Prevention through antimicrobial feed additives remain common but have not proven to eliminate liver abscess incidence. Nutritional management is a more feasible preventative option but can be costly. The objective of this study was to use quantitative genetics to produce a preliminary heritability estimate for liver abscess incidence and severity in beef on dairy heifers. Data were collected on 1,860 beef on dairy heifers fed and harvested in Kansas. Animals without sire information were removed, leaving 1,492 heifers in the data set. The 1,492 heifers had sire information, but none had dam information. A 3-generation pedigree was utilized based on the sire parentage information. Liver abscess scores were assigned at slaughter. Liver abscess scores were separated into three types: numerical 0 to 3 range representing unique scores, binary representing no abscess versus abscess, and binary representing no abscess versus severe abscess score of "A+". Nine mixed effect sire models were run to estimate heritability. Models 1, 2, and 3 were continuous univariate models with the numerical scoring system. Models 4, 5, and 6 were binary threshold models with the no abscess versus abscess scoring system. Models 7, 8, and 9 were binary threshold models with no abscess versus severe abscess. Models 1, 4, and 7 had no sires removed from the data set. Models 2, 5, and 8 used a data set with heifer records removed if they were from sires with less than ten progeny. Models 3, 6, and 9 used a data set with heifer records from sires with between 100 and 200 progeny in the complete data set. Heritability estimates from a sire model for

Models 1, 4, and 7 ranged from 4.26 x $10^{-8}$ to 1.06 x $10^{-7}$. Heritability estimates from a sire model for Models 2, 5, and 8 ranged from 4.90 x $10^{-8}$ to 4.61 x $10^{-7}$. Heritability estimates from a sire model for Models 3, 6, and 9 ranged from 1.01 x $10^{-7}$ to 2.88 x $10^{-3}$. All estimates were equivalent to zero. The preliminary heritability estimates of zero revealed no genetic component to liver abscess incidence or severity in this data set.

## INTRODUCTION

Liver abscesses continue to impact economics, performance, and animal welfare in the beef industry. Liver abscesses in harvested cattle have been of concern to the industry since the 1800s, with reports of liver abscesses and a potential theory for their cause as early as 1891 (McFadyean, 1891). The accepted pathogenesis of liver abscesses in cattle since the 1950s was the rumenitis-liver abscess complex, where acidosis leads to rumenitis and then to liver abscesses (Jensen et al., 1954a). When cattle are fed high levels of grains without proper transition, the lack of structure in the grains allows for the microorganisms in the rumen to begin fermentation of the carbohydrates more rapidly (Hernández et al., 2014). The fermentation process produces volatile fatty acids (VFA) faster than they can be absorbed, decreased buffer levels, and eradication of pH-sensitive bacteria contribute to the low pH of the rumen. The pH of the rumen during acidosis is less than 5.5 (Nagaraja and Titgemeyer, 2007; Owens et al., 1998; Hernández et al., 2014). Rumenitis can be caused by this acidic environment, resulting in inflammation and ulceration of the ruminal epithelium. Bacteria and endotoxins naturally occurring in the rumen are then able to exit the irritated rumen to enter the portal vein and splanchnic circulatory system (Krause and Oetzel, 2006; Oetzel, 2017; Aschenbach et al., 2019; Plaizier et al., 2018). When the

blood containing the rumen bacteria enters the liver, abscesses form due to the presence of the bacteria in the liver (Nakajima et al., 1986). Adhesion of the liver to nearby organs, such as the diaphragm and gastrointestinal tract, can occur due to the fibrinous inflammation from the abscesses (Jensen et al., 1954b; Nagaraja and Titgemeyer, 2007).

The primary cause of diagnostic difficulty pre-harvest is the lack of clinical signs in the live animal. While diagnosis and treatment of liver abscesses prior to slaughter can be accomplished, the asymptomatic nature of liver abscesses prevents animal caretakers from identifying diseased animals (Doré et al., 2007; Braun, 2009). Rather than diagnosis and treatment, focus has turned to prevention. At the time of writing, the most common prevention tool in conventional operations is antimicrobial feed additives. Tylosin phosphate is one of the most common and effective antimicrobial feed additives. The feeding of tylosin significantly lowers liver abscess incidence and severity, but does not eliminate abscesses (Theurer and Amachawadi, 2022). Nutritional management is another successful tool to decrease acidosis prevalence through proper diet, meal size, and transition to grain (González et al., 2012). Antimicrobial feed additives and proper nutritional management are the primary prevention methods used by producers.

To determine the feasibility of genetic selection as a tool for reducing incidence of liver abscesses, Keele et al. (2016) performed a genome-wide association study on beef cattle, finding indications of a potential genetic component to liver abscesses. Thirty-five single nucleotide polymorphisms (SNP) were identified in association with liver abscesses indicating genetic selection may be possible (Keele et al., 2016).

While there has been identification of genetic components of liver abscesses through molecular genetic techniques, there has not been published investigation of a genetic component

in liver abscesses through quantitative approaches. The objectives of this study were to use quantitative genetics to produce a preliminary heritability estimate for liver abscess incidence and severity in beef on dairy heifers.

## MATERIALS AND METHODS

Data were collected on 1,860 heifers finished at a feedlot in Kansas. Animals without known sires were removed from the dataset, leaving 1,492 heifers for evaluation. Individuals were harvested during the 2022 to 2023 calendar years. At the feedlot, animals had phenotypes collected for feedlot in-weight in kg and number of treatments for bovine respiratory disease (BRD). The animals were separated into different kill lots and received different treatments at the feedlot. The treatments were not of statistical interest in this study but were part of a larger study evaluating the efficacy of different treatments to mitigate liver abscess incidence. To account for the variation due to treatment, treatment was included in the contemporary group as this accounts for additional variation introduced into the data due to the different treatment categories. At harvest in Kansas, liver abscess scores were assigned using the Elanco Liver Scoring System (Elanco Animal Health, Greenfield, IN) as described in Table 4.1. All animals received liver abscess score phenotypes.

Table 4.1 Liver abscess scores and descriptions based on Elanco Liver Scoring System (Elanco Animal Health, Greenfield, IN) with combined score of "A+" that includes scores of "A+AD" and "A+O".

| Liver Abscess Score | Description |
|---|---|
| 0 | No abscess present. |
| A- | One or two abscesses that are less than 2.5 cm in diameter. |
| A | Numerous abscesses that are less than 2.5 cm in diameter OR one or two abscesses greater than 2.5 cm in diameter. |
| A+ | Numerous abscesses greater than 2.5 cm in diameter OR Liver adhered to a nearby organ OR Liver abscess ruptured |

Liver abscess scores of "A+", "A+AD", and "A+O" were combined to form a more general "A+" category that represented the more severe presence of an abscess. Each score was given a number to represent liver abscess scores continuously and numerically. The transformation was "0" = 0, "A-" = 1, "A" = 2, "A+" = 3. To represent liver abscess scores as a binary score of non-abscessed and abscessed, scores of "0" kept as 0, and scores of "A-", "A", and "A+" were renumbered to 1. To represent liver abscess scores as a binary score of non-abscess and severe abscess, scores of "0" were kept as 0, scores of "A+" were renumbered to 1, and all remaining scores were removed from the data set.

The heifers in the data set had a verified sire; however, none had dam information. A three-generation sire pedigree was utilized. Forty-eight unique animals were present in the three-generation sire pedigree. There were 19 unique sires of the heifers in the data set. All 19 sires had recorded sires and grandsires. Only 3 sires also had great grandsires. There was no dam information in the pedigree. A subset of the data was utilized when removing heifers from sires with less than ten progeny in the data set. Following removal, 9 sires remained with a pedigree of 29 unique animals. The subset of data had 1,448 unique heifers and observations. Another subset of the data was utilized using only sires that had more than 100 but less than 200 progeny in the complete data set. This subset of data had 482 unique heifers and observations.

Six data sets were used in this study. The data set that included all animals and liver abscess scores was identified as “ALL” in this study. The subset that removed heifers from sires with less than ten progeny was identified as “ALLS1” in this study. The subset that included only heifers from sires with 100 to 200 progeny was identified as “ALLS2”, this included only four sires. The data set that included all animals and only liver abscess scores of “0” or “A+” was identified as “EXT” in this study. The subset that removed heifers from sires with less than ten progeny was identified as “EXTS1” in this study. The subset that included only heifers from sires with 100 to 200 progeny in the complete data set was identified as “EXTS2”, this included only four sires.

Mixed effect model selection was conducted using R (R Core Team, 2023), RStudio (Posit team, 2024), the lme4 package (Bates et al., 2015), and the performance package (Lüdecke et al., 2021). The response variable was liver abscess score on a continuous numerical scale. Contemporary group was a categorical variable. Age in days, number of BRD treatments, and feedlot in-weight were continuous variables. The “Base” model had fixed effects of contemporary group and age in days, with a random effect of sire. The “BRD” model had fixed effects of contemporary group, age in days, and number of BRD treatments, with a random effect of sire. The “Weight” model had fixed effects of contemporary group, age in days, and feedlot in-weight, with a random effect of sire. The “Full” model had fixed effects of contemporary group, age in days, number of BRD treatments, and feedlot in-weight, with a random effect of sire. Using AIC values, partial F-test, and visual assessment of assumptions of linearity, homogeneity of variance, and normality of residuals, the model of best fit was determined. Contemporary group was a combination of kill lot and treatment.

All heritability estimates were conducted using ASREML 3.0 (VSN International, Ltd., Hemel Hempstead, UK). The univariate sire models used for Model 1, Model 2, and Model 3 are presented in matrix form below.

$$\boldsymbol{Y} = \boldsymbol{X\beta} + \boldsymbol{Zs} + \boldsymbol{e}$$

In the matrix form, $\boldsymbol{Y}$ was a vector of observations for liver abscess score, $\boldsymbol{X}$ was an incidence matrix relating observations to the fixed effects, $\boldsymbol{\beta}$ was a vector of fixed effects for contemporary group and age, $\boldsymbol{Z}$ was an incidence matrix relating observations to additive genetic effects, $s$ was a vector of sire genetic effects, and $e$ was a vector of random residuals specific to each observation. Random effects were assumed to have means of 0 and variances represented as $var(\boldsymbol{u}) = \boldsymbol{A}\sigma_u^2$ and $var(\boldsymbol{e}) = \boldsymbol{I}\sigma_e^2$, where $\boldsymbol{A}$ was Wright's numerator relationship matrix, and $\boldsymbol{I}$ was an identity matrix whose order is equal to the number of observations in $\boldsymbol{Y}$. The threshold sire models for the underlying distribution of the score were as described for the univariate sire model. Contemporary groups that included only one animal were removed from the data set.

Nine models were run using the fixed and random effects from the best-fit model. The models are described in Table 4.2.

Table 4.2 Model descriptions of the six utilized in estimating heritability of liver abscesses.

| Model Number | Scoring System [1] | Data Set [2] |
|---|---|---|
| 1 | 0, A-, A, A+ | ALL (n = 1,492) |
| 2 | 0, A-, A, A+ | ALLS1 (n = 1,448) |
| 3 | 0, A-, A, A+ | ALLS2 (n = 482) |
| 4 | No abscess vs. Abscess | ALL (n = 1,492) |
| 5 | No abscess vs. Abscess | ALLS1 (n = 1,448) |
| 6 | No abscess vs. Abscess | ALLS2 (n = 482) |
| 7 | No abscess vs. Severe abscess | EXT (n = 1,254) |
| 8 | No abscess vs. Severe abscess | EXTS1 (n = 1,217) |
| 9 | No abscess vs. Severe abscess | EXTS2 (n = 401) |

[1] Liver abscess transformed scoring system
[2] Data set used. ALL = Complete data set, ALLS1 = Complete data set with records from heifers from sires with less than 10 progeny removed, ALLS2 = Complete data set with records from heifers from sires with more than 100 and less than 200 progeny, EXT = Data set with normal ("0") and severe scores ("A+"), EXTS1 = Data set with normal ("0") and severe scores ("A+") with records from heifers from sires with less than 10 progeny removed, EXTS2 = Data set with normal ("0") and severe scores ("A+") with records from heifers from sires with more than 100 and less than 200 progeny in complete data set

## RESULTS AND DISCUSSION

In model development, the model with the lowest AIC value was the "Base model" with fixed effects of contemporary group and age and the random effect of sire (*AIC* = 4996.07). The next lowest AIC value was that of the BRD model with fixed effects of contemporary group, age, and number of BRD treatments, with random effect of sire (*AIC* = 4998.14). There was no improvement in goodness of fit when in-weight or in-weight and BRD treatments were included. A partial F-test was conducted to further determine whether the Base model or the BRD model should be utilized. There was evidence of a linear relationship between average liver abscess score and BRD treatment number in a model that already contained contemporary group and age in days with a random sire effect ($P < 0.05$).

Visual assessment of assumptions of linearity, homogeneity of variance, and assumption of normality was done using a residuals versus fitted values plot and a sample quantile deviations versus standard normal distribution quantiles plot. Figure 4.1 contains the plots for the Base model, with A referencing the plot for assumption of linearity and homogeneity of variance and B referencing the plot for assumption of the normality of residuals. In Figure 4.1 A, there is a relatively flat trend in the residuals versus fitted values plot, so the assessment was no violation of linearity. The gray shaded area is the confidence intervals for the reference line. In Figure 4.1 A, there is fairly even spread of residuals across all fitted values, so the assessment was no violation of homogeneity of variance. In Figure 4.1 B, there are deviations at a high value, with deviations above 1.0, and a distinct trend in the residuals, so there was a violation of the normality of residuals assumption. Figure 4.1 contains the plots for the BRD model, with A referencing the plot for assumption of linearity and homogeneity of variance and B referencing the plot for assumption of the normality of residuals. For the BRD model, Figure 4.2 A also reveals a relatively flat trend in the residuals versus fitted values plot with an even spread of residuals, so the assessment was that there were no violation of linearity or homogeneity of variance assumptions. In Figure 4.2 B, there are also high deviations above 1.0 with a distinct trend; however, the deviations are not as high as those in B, so the assessment was that there was a lesser violation of the normality of residuals assumption for the BRD model than the Base model.

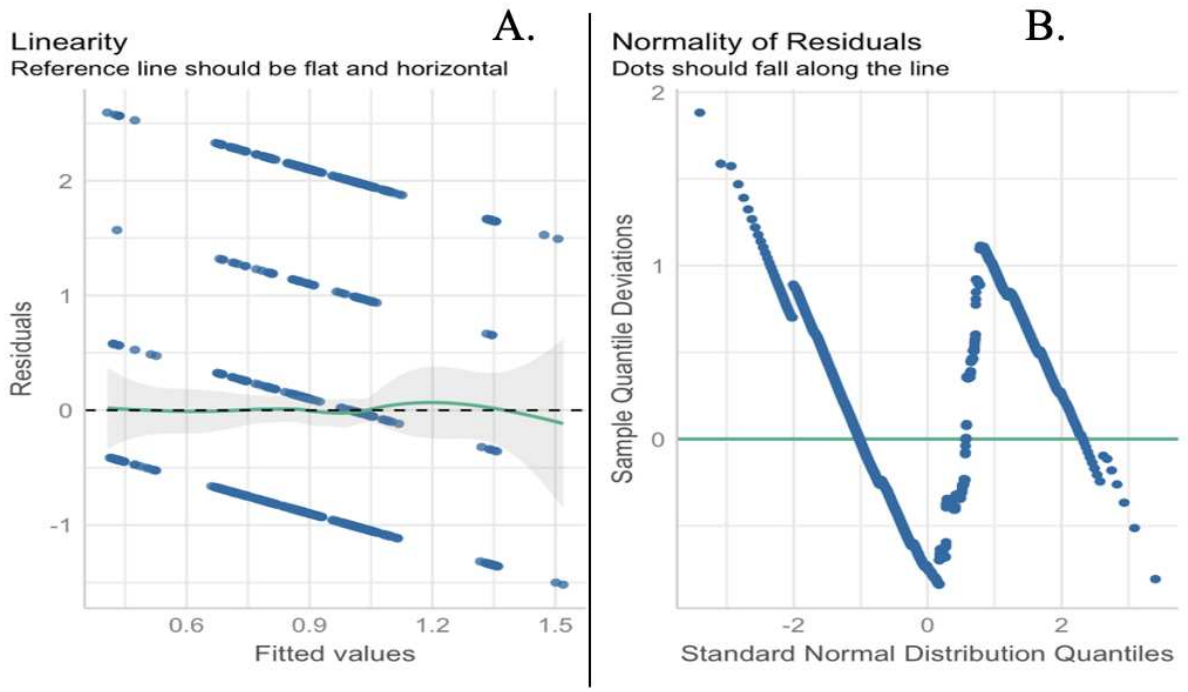


Figure 4.1 For Base Model. A. Residuals versus fitted values plot for linearity and homogeneity of variance assumptions. B. Sample quantile deviations versus standard normal distribution quantiles plot for assumption of normality of residuals.

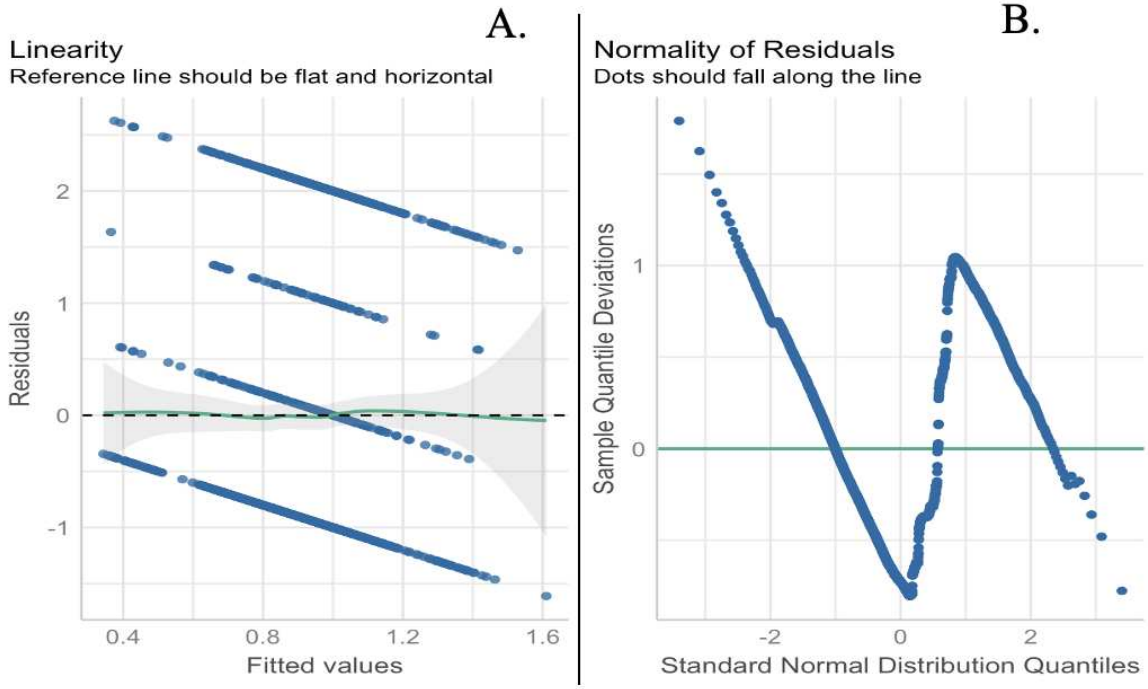


Figure 4.2 For BRD Model. A. Residuals versus fitted values plot for linearity and homogeneity of variance assumptions. B. Sample quantile deviations versus standard normal distribution quantiles plot for assumption of normality of residuals.

The best fit model was the BRD model. While the Base model had a lower AIC value ($AIC = 4996.07$), the significant partial F-test for number of BRD treatments inclusion as a predictor variable ($P < 0.05$), and the decrease in normality of residuals violation indicate the BRD model may be a better fit. The BRD model is the model used throughout this study. When fit,contemporary group and age in days were not significant in the overall model ($P > 0.05$). Contemporary group and age in days were still included in the model to account for environmental differences between animals. Number of BRD treatments was significant in the overall model ($P < 0.05$).

The point estimate of BRD treatment on liver abscess score was significant and positive, indicating an increase of 0.05 in liver abscess score with every 1 treatment increase in BRD treatments. All sires in this study had at least one progeny with a non-zero number of BRD treatments. In Rezac et al. (2014), out of all the cattle with severe liver abscesses, 28.3% also had mild lung lesions, and 14.9% had severe lung lesions, indicative of BRD at some stage during their life. Grimes (2022) and Herrick (2018) both identified significant relationships between BRD and liver abscesses in cattle, with more lung lesions associated with more liver abscesses. A relationship may exist between liver abscesses and BRD because immune and physiological stressors, caused by either the liver abscess or BRD lead to higher susceptibility to the other disease. Through discussions with multiple producers, anecdotally, animals that have had more experiences of BRD having decreased incidence of liver abscess. Those claims are not consistent with what has been reported in the literature, but they do provide an alternate hypothesis that could be further investigated. More formal studies are required to solidify the relationship between liver abscesses and BRD, potential causes, and why some producers see increased BRD incidence associated with decreased liver abscess incidence.

In the complete data set with no sire removal (ALL), there were 1,492 animals, with no observations missing. All liver abscess scores were represented in ALL. In ALL, the distribution of liver abscess scores is shown in Figure 4.3. The A+ category had a total of 352 observations, with 232 being A+AD, 14 being A+O, and 106 being an actual score of A+. Summary statistics for the ALL data set, including binary and numeric liver abscess scores, number of BRD treatments, and age in days, are shown in Table 4.3.

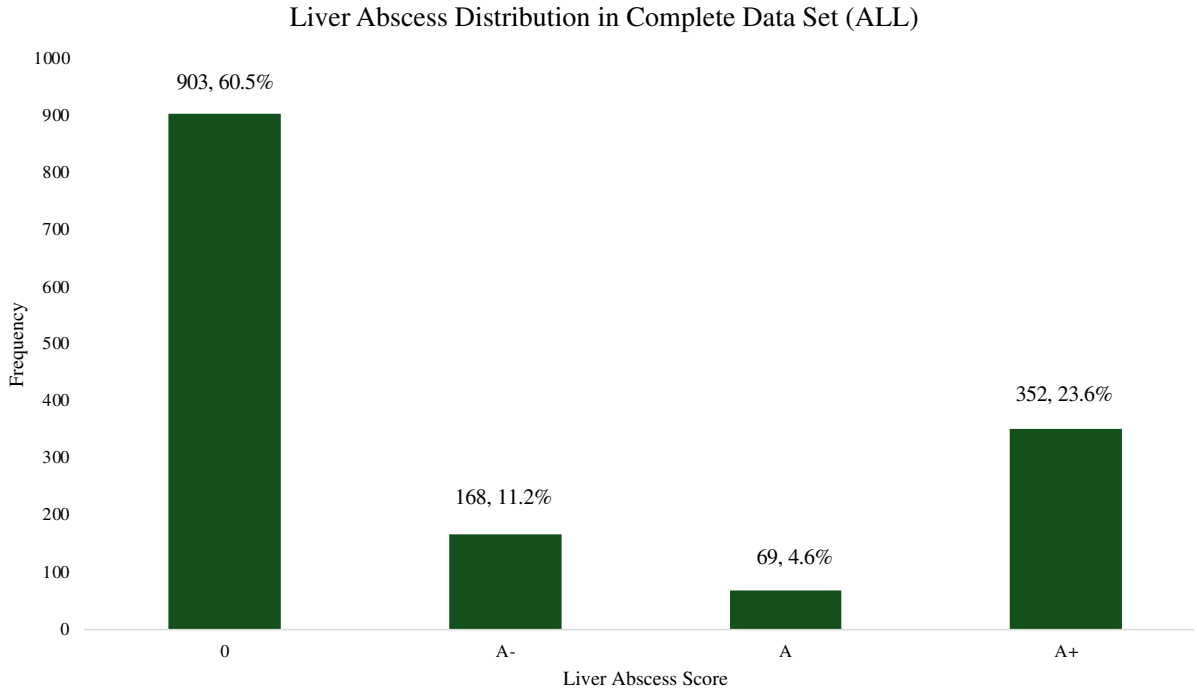


Figure 4.3 Liver abscess score distribution for the complete data set with no sire removals (ALL).

Table 4.3 Number of observations (N), mean, standard deviation, minimum, and maximum for number of BRD treatments and age in days for the data set including all scores (ALL).

| | N | Mean | Standard Deviation | Minimum | Maximum |
|---|---|---|---|---|---|
| Number of BRD Treatments | 1492 | 1.04 | 1.43 | 0 | 10 |
| Age in Days | 1492 | 520.75 | 35.28 | 404 | 676 |

In the data set for extreme scores (0 vs. A+) with no sire removal (EXT), there were 1,254 animals possessing known observations. Only scores of 0 and A+ (inclusive of A+AD and

A+O) were represented in this data set. There were 902 animals with scores of 0 and 352 animals with scores of A+, as illustrated in Figure 4.4. Summary statistics for the EXT data set, including binary liver abscess scores, number of BRD treatments, and age in days, are shown in Table 4.4. Notable differences from the summary statistics for ALL were that ALL has a higher mean and standard deviation for number of BRD treatments, higher standard deviation for age in days, lower mean for age in days, and lower minimum age in days value than EXT.

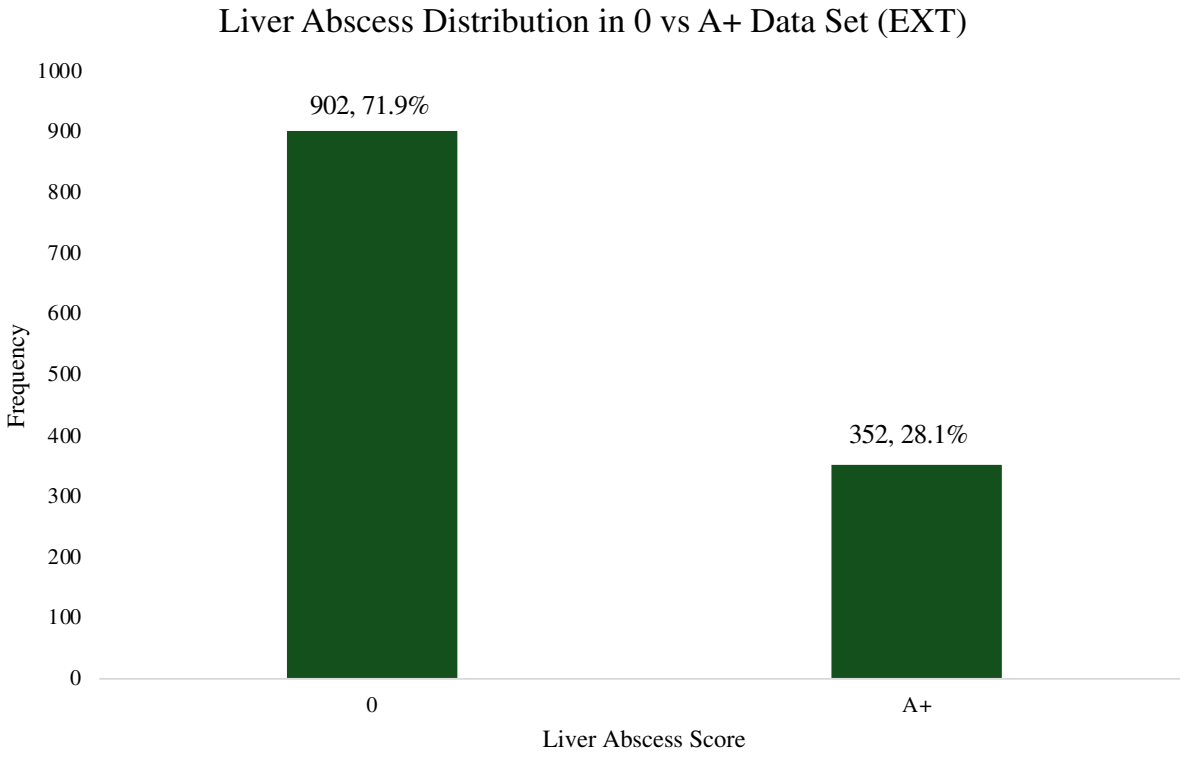


Figure 4.4 Liver abscess score distribution for the data set with only scores of "0" or "A+" (EXT).

Table 4.4 Number of observations (N), mean, standard deviation, minimum, and maximum for number of BRD treatments and age in days for the data set only including scores of 0 and A+ (EXT).

| | N | Mean | Standard Deviation | Minimum | Maximum |
|---|---|---|---|---|---|
| Number of BRD Treatments | 1254 | 1.02 | 1.42 | 0 | 10 |
| Age in Days | 1254 | 520.93 | 35.07 | 407 | 676 |

The distribution of numeric liver abscess scores in ALL split by sire is illustrated in Figure 4.5 A. For the 1,492 animals in the data set, they all come from one of 19 unique sires. Figure 4.5 B and C are the distributions of liver abscess scores in the ALLS1 and ALLS2 data sets, respectively. Figure 4.6 A, B, and C follow the same organization of subsets, but applied to the EXT data set illustrating the binary 0 vs A+ scores distribution in EXT. The 1,254 animals in EXT also come from the same 19 unique sires as in ALL. The sire IDs have been relabeled to A through S. The inability to visualize the frequencies for certain sires due to the lack of progeny in the data sets emphasized the lack of sire variation and unequal spread. Sire E had 530 progeny in the data set, while others had one progeny in the data set. ALLS2 and EXTS2 only included 4 sires. In ALLS2, the number of progeny for each sire ranged from 101 to 151. The same sires were used in EXTS2, but then had a range of progeny of 86 to 123. The purpose was to identify sires with similar progeny amounts, so even though the range went below 100, they were still kept in EXTS2.

The uneven spread of sires also infiltrates contemporary groups. There were 30 unique contemporary groups in the ALL data set. The range in number of heifers in each contemporary group is 2 to 67, with an average of 50 heifers. The range in number of sires represented in each contemporary group is 2 to 10, with an average of 7 represented sires. No contemporary group had all sires represented. The number of contemporary groups a sire was in ranged from 1 to 27,

with an average of 11 contemporary groups. Only one sire was in a single contemporary group due to them having only one offspring in the data set. This sire and their progeny were kept in the ALL and EXT data sets. Having varied levels of sire representation in each contemporary group and the sires not consistently showing up in the same contemporary groups were consequences of the limited number of unique sires and the uneven number of progeny per sire.

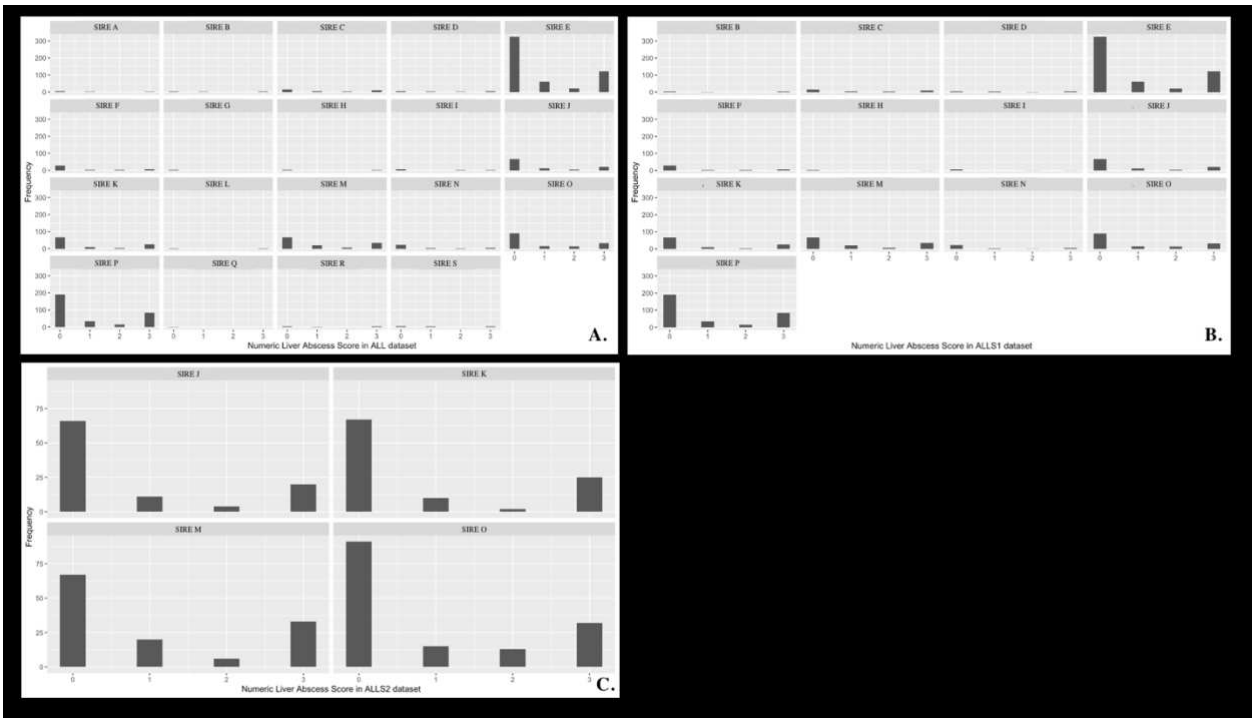


Figure 4.5 Distributions of liver abscess score by sire in complete data set. A. All scores with no sires removed (ALL). B. All scores with sires removed (ALLS1). C. All scores with four sires (ALLS2).

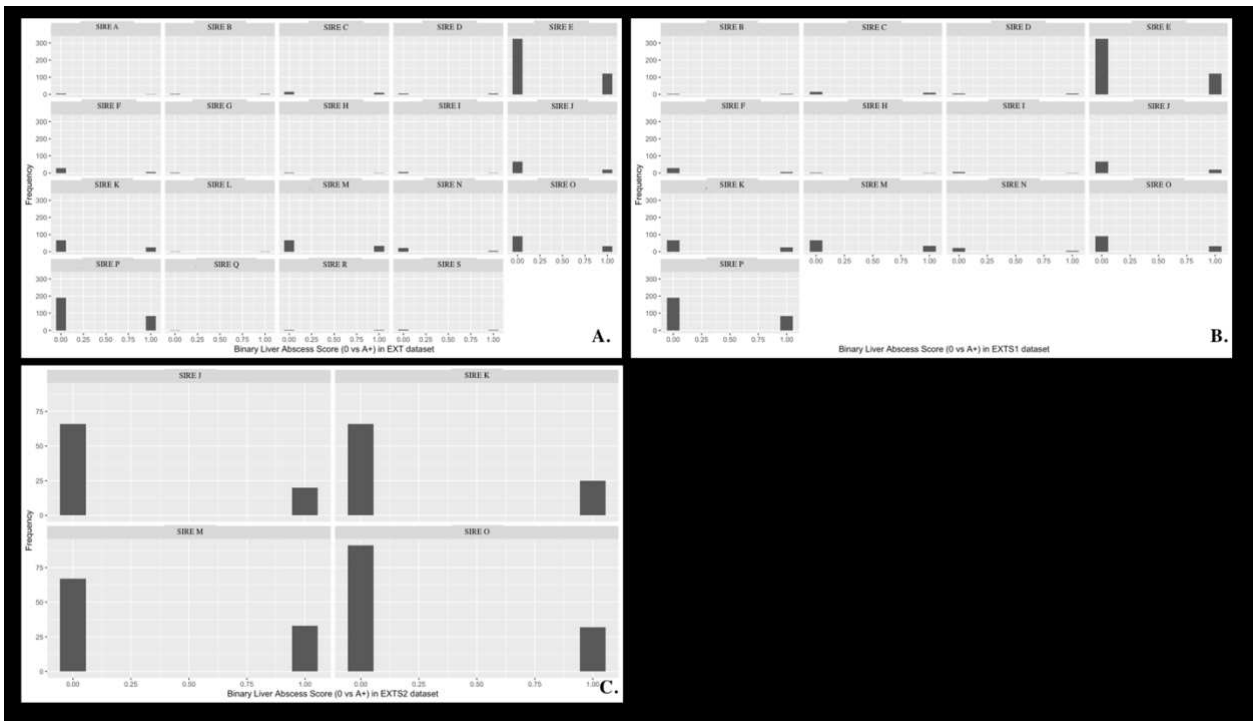


Figure 4.6 Distributions of liver abscess score by sire in extreme data set. A. Extreme scores with no sires removed (EXT). B. Extreme scores with sires removed (EXTS1). C. Extreme scores with only sires with 100 to 200 progeny (EXTS2).

The mixed effect sire models used to estimate heritability using continuous, numeric liver abscess scores were Models 1, 2, and 3. The genetic variance, phenotypic variance, and heritability are reported in Table 4.5. The heritability estimates for Model 1, Model 2, and Model 3 were $4.26 \times 10^{-8}$, $4.90 \times 10^{-8}$, and $1.01 \times 10^{-7}$ respectively. All phenotypic variances were similar, with Model 3 having a slightly larger genetic variance and heritability estimate. While there was some difference in the genetic variance and heritability values, both were effectively 0. Using a continuous mixed effect sire model, the liver abscess score was not heritable.

Table 4.5 Genetic variance, phenotypic variance, and heritability estimate for data set with all liver abscess scores.

| | Model 1 | Model 2 | Model 3 |
|---|---|---|---|
| Genetic Variance | 0.000000067 | 0.000000078 | 0.00000015 |
| Residual Variance | 1.58462 | 1.58251 | 1.51723 |
| Phenotypic Variance | 1.58462007 | 1.58251008 | 1.51723015 |
| Heritability | 4.26E-08 | 4.90E-08 | 1.01E-07 |

Model 1 = Continuous model from the complete data set (ALL)
Model 2 = Continuous model from the sire-removed complete data set (ALLS1)
Model 3 = Continuous model from the four-sire complete data set (ALLS2)
Heritability = genetic variance / phenotypic variance

The mixed effect sire models used to estimate heritability using a threshold model indicating no abscess or abscess were Models 4, 5, and 6. Model 4 used the ALL data set. Model 5 used the ALLS1 data set. Model 6 used the ALLS2 data set. The phenotypic and genetic variances and heritability for Models 4 and 5 were similar, as shown in Table 4.6. Model 6 had the highest heritability estimate. The heritability estimates from Models 4, 5, and 6 were $1.06 \times 10^{-7}$, $4.61 \times 10^{-7}$, and $2.88 \times 10^{-3}$ respectively. All were still effectively 0. While the heritability estimate from Models 6 was larger than those produced in Models 1 to 5, liver abscess incidence was still not heritable.

Table 4.6 Genetic variance, phenotypic variance, and heritability estimate for data set with all liver abscess scores.

| | Model 4 | Model 5 | Model 6 |
|---|---|---|---|
| Genetic Variance | 0.00000011 | 0.00000046 | 0.0028837 |
| Residual Variance | 1 | 1 | 1 |
| Phenotypic Variance | 1.00000011 | 1.00000046 | 1.0028837 |
| Heritability | 1.06E-07 | 4.61E-07 | 2.88E-03 |

Model 4 = Threshold model from complete data set (ALL)
Model 5 = Threshold model from the sire-removed complete data set (ALLS1)
Model 6 = Threshold model from the four-sire complete data set (ALLS2)
Heritability = genetic variance / phenotypic variance

The mixed effect sire models used to estimate heritability using a threshold model indicating no abscess or a score of A+ were Models 7, 8, and 9. Model 7 used the EXT data set. Model 8 used the EXTS1 data set. Model 9 used the EXTS2 data set. The variance and heritability estimates were similar for Models 7 and 8, but Model 9 had a higher genetic variance, phenotypic variance, and heritability, as shown in Table 4.7. The heritability estimates for Model 7, Model 8, and Model 9 were $4.91 \times 10^{-8}$, $5.25 \times 10^{-8}$, and $3.85 \times 10^{-7}$. All were still effectively 0, indicating that liver abscess scores of 0 versus A+ were not heritable.

Table 4.7 Genetic variance, phenotypic variance, and heritability for data set with only scores of 0 and A+.

| | Model 7 | Model 8 | Model 9 |
|---|---|---|---|
| Genetic Variance | 0.000000049 | 0.000000053 | 0.000000385 |
| Residual Variance | 1 | 1 | 1 |
| Phenotypic Variance | 1.000000049 | 1.000000053 | 1.000000385 |
| Heritability | 4.91E-08 | 5.25E-08 | 3.85E-07 |

Model 7 = Threshold model from 0 vs. A+ data set (EXT)
Model 8 = Threshold model from 0 vs. A+ sire-removed data set (EXTS1)
Model 6 = Threshold model from the 0 vs. A+ four-sire data set (EXTS2)
Heritability = genetic variance / phenotypic variance

Overall, all heritability estimates regarding liver abscess incidence were zero. The range in heritability estimates for all models conducted using information from all 19 unique sires was $4.26 \times 10^{-8}$ to $1.06 \times 10^{-7}$. The range in heritability estimates for all models conducted using information from heifers from sires with ten or more progeny represented in the data set was $4.90 \times 10^{-8}$ to $4.61 \times 10^{-7}$. The range in heritability estimates for all models conducted using information from heifers from the four sires with 100 to 200 progeny in the complete data set was $1.01 \times 10^{-7}$ to $2.88 \times 10^{-3}$. The model type with the highest heritability estimates were the threshold binary models; however, they were still not different than zero. The data set with the highest heritability estimates were the ALLS2 and EXTS2 data sets where it included only heifers from sires with similar ranges of progeny in the data set. While Model 6 was the highest heritability and the closest to 0, it was still essentially zero.

The nature of liver abscesses presented a limitation in this study. Liver abscesses typically are not diagnosed prior to slaughter. Berry et al. (2011) identified that a common limitation to disease trait genetic evaluation is those measured later in life. Late in life measurement leads to a longer wait time before performance can be confirmed and then included in further genetic evaluations (Berry et al., 2011). Since the animal's disease status is measured at a fixed point in time, there is an inherent lack of data regarding healed abscesses, how often abscesses formed, when the current abscesses first formed, if the abscess would have gotten more severe over time, and other information about the animal's liver status during their entire lifespan. While not feasible in production settings, if liver abscesses could be measured repeatedly using diagnostic tools while the animal was alive, more information could be collected to gain a better understanding of the individual and their pathological responses to liver abscesses. More observations and pedigree information are necessary in providing higher

accuracy heritability estimates. Further research is crucial in the determination of genetic selection feasibility for liver abscesses in beef on dairy heifers

# CONCLUSION

Liver abscess scores and incidence were not heritable in this preliminary study. While no other published study at this time has reported a heritability estimate for liver abscesses, Keele et al. (2016) does show indications of a genetic component on a genomic level. Differences in the structure of these studies may play a role in the findings. The Keele et al. (2016) study had 2,304 samples from beef cattle going through a commercial facility over a week and followed by a genome-wide association (Keele et al., 2016). Our study had, at most, 1,492 beef on dairy animals from the same feedlot and looked solely at phenotypic data with only sire information. The animals from the same originating producer contributed to the limitations in sire variation in this study. The difference between the results of these studies indicates that there is a need for further exploration of liver abscess genetics to determine if there is a non-zero genetic component.

The lack of pedigree information remains the largest limitation of this study. Limited numbers of unique beef sires used on many dairy cows to produce beef on dairy animals are common in current beef-on-dairy systems. If dam information could be collected, the pedigree would account for more relationships between the animals of this data set and would allow for a higher accuracy heritability estimate. A more even distribution of sires throughout contemporary groups and in number of progeny represented in the data set would also benefit this study. We saw higher estimates using sires with similar ranges of progeny, however, that then limited the

number of records included in the analysis. With the utilization of more observations and animals with complete pedigree information, further investigation of the genetic components of liver abscesses is required. Continued investigation on non-genetic prevention tools for liver abscesses is also recommended.

The results of this study indicate no genetic component linked to liver abscess phenotype. Genetic selection feasibility must be determined following the continued study of liver abscess heritability.

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# CHAPTER 5: CONCLUSIONS

In the first study, beef on dairy heifer carcass traits were evaluated to identify associations with liver abscess scores. Hot carcass weight, fat thickness, rib eye area, marbling score, calculated visual yield grade and heart score were analyzed as the carcass traits of interest. Liver abscess scores were included as independent variables using all six scores present in the Elanco Scoring System (Elanco Animal Health, Greenfield, IN) or utilizing four scores of "0", "A-", "A", and a combined severe abscess score of "A+" that included all scores of "A+", "A+AD", and "A+O". Using the six-score system, back fat thickness and yield grade were significantly higher for animals with scores of "A+O" compared to those with scores of "A+". Yield grade also had a significant increase for animals with scores of "A+O" compared to those with scores of "A+". While not significant, there were tendency differences in FT between animals with scores of "A" and "A+AD" compared to "A+O" ($0.05 \leq P < 0.10$). The least-squares means for fat thickness were 1.59 cm ± 0.06 and 1.94 cm ± 0.12 for animals with scores of "A+" and "A+O", respectively ($P < 0.05$). The least-squares means for yield grade were 3.20 ± 0.09, 3.20 ± 0.08, and 3.75 ± 0.19 for animals with scores of "A+", "A+AD", and "A+O", respectively ($P < 0.05$). Using the four-score system, hot carcass weight was statistically lower for animals with scores of "A+" compared to those with no liver abscesses. Hot carcass weight least-squares mean, and standard error was 396 kg ± 2.63 for animals with no liver abscesses and 391 kg ± 2.92 for animals in the grouped severe liver abscess score ($P < 0.05$). While the animals used in both scoring systems were the same, the sample size did decrease for the severe abscess scores when they were analyzed individually instead of as a group. The severe grouping had 441

animals, but those were split further into 138, 287, and 16 animals for scores of “A+”, “A+AD”, and “A+O”, respectively. This could have contributed to differences in significant findings.

A decrease in hot carcass weight in animals with severe abscesses may be because of an animal not eating as much, complications in nutrient metabolism in the liver because of decreased functionality with severe abscesses and adhesion, or energy partitioned to the immune system response rather than muscle and fat synthesis. Having animals with decreased hot carcass weights is not desirable in American beef production because it decreases the amount of saleable product. The reasons for an increase in fat thickness and yield grade in animals with severe abscesses relates back to those originating for sickness. Causes could be due to lack of activity, decreased feed intake, or physiological responses to stress leading to increased fat deposition. Higher yield grades and increased fat thickness are not desirable because there is more trim required and a lower ratio of muscle to fat.

Similar results have been identified in comparable studies, but even within those studies, results differ between data sets with different management styles and breeds. Differences in these results may indicate a potential underlying unidentified variable that contributes to these differences more than liver abscess severity does. Further studies evaluating effects with a grouped severe abscess score compared to individual scores would assist in determining the true relationship between scores and performance.

The second study focused on estimating the heritability of liver abscesses in beef on dairy heifers using different modeling techniques and sire inclusions. Models 1, 2, and 3 evaluated liver abscesses on a continuous basis, with “A+” being a grouped severe abscess score including “A+”, “A+AD”, and “A+O”. Model 1 included all data, Model 2 only included data from heifers who came from sires that had ten or more progeny in the data set, and Model 3 only included

data from heifers who came from sires that had 100 to 200 progeny in the complete data set. Heritability estimates for Models 1, 2, and 3 were $4.26 \times 10^{-8}$, $4.90 \times 10^{-8}$, and $1.01 \times 10^{-7}$ respectively. Models 4, 5, and 6 evaluated liver abscesses on a threshold basis with a binary score of no abscess versus abscessed. Model 4 included all data, Model 5 only included data from heifers who came from sires that had ten or more progeny in the data set, and Model 6 only included data from heifers who came from sires that had 100 to 200 progeny in the complete data set. Heritability estimates for Models 4, 5, and 6 were $1.06 \times 10^{-7}$, $4.61 \times 10^{-7}$, and $2.88 \times 10^{-3}$ respectively. Models 7, 8, and 9 evaluated liver abscesses on a threshold basis with a binary score of no abscess versus severe abscess score that combined "A+", "A+AD", and "A+O". Model 7 included all data, Model 8 only included data from heifers who came from sires that had ten or more progeny in the data set, and Model 9 only included data from heifers who came from sires that had 100 to 200 progeny in the complete data set. Heritability estimates for Models 7, 8, and 9 were $4.91 \times 10^{-8}$, $5.25 \times 10^{-8}$, and $3.85 \times 10^{-7}$ respectively. All estimates are not different from zero.

The most prominent limitation with estimating heritability in this data set was the lack of number of sires and unequal spread of progeny across sires. With 1,492 animals, only 19 sires were represented. No dam information was available for these heifers, which prevented the inclusion of many relationships between the heifers. Within the 19 sires, they were unevenly spread throughout the sample population and contemporary groups, with progeny per sire ranging from 1 to 530. To attempt and correct for the wide range of progeny per sire, Models 2, 3, 5, 6, 8, and 9 were conducted. While heritability estimates were slightly higher, they were still zero. The highest estimates came from the data set that included only heifers who came from sires that had 100 to 200 progeny in the complete data set. The sire variation problem was due to

standard beef on dairy production system management, which works well in the industry but was not ideal for the research goals of this study.

The estimate of zero heritability for liver abscesses in this data set does not inherently eliminate genetic selection from being possible. Genetic variance of liver abscess has been identified in beef cattle using genomics and suggests the use of genomics compared to only phenotypic observations might allow for higher accuracy. To better estimate heritability using phenotypes and pedigree information, additional phenotypes from a larger number of cattle from many additional sires are required. While the results of this study reveal very low heritability estimates, this study was preliminary, with less information than desirable, and should be repeated before eliminating genetic selection as a viable prevention method. Future studies on liver abscesses are required to finalize a decision on genetic selection, as even a low, non-zero heritability estimate can be utilized for genetic selection. Regardless of future estimates, environmental factors continue to be the primary influence on liver abscesses.

Overall, the true effects of liver abscesses and their genetic components in cattle vary within this study and compared to similar studies. Further research is necessary to provide confirmation and further knowledge of the impacts of liver abscesses and their genetic components in beef on dairy cattle.