THESIS

GENETICS OF BOVINE RESPIRATORY DISEASE IN FEEDLOT CATTLE

Submitted by

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WE HEREBY RECOMMEND THAT THE THESIS PREPARED UNDER OUR SUPERVISION BY CHASE M. McALLISTER ENTITLED GENETICS OF BOVINE RESPIRATORY DISEASE IN FEEDLOT CATTLE BE ACCEPTED AS FULFILLING IN PART REQUIREMENTS FOR THE DEGREE OF MASTER OF SCIENCE.

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ABSTRACT OF THESIS

GENETICS OF BOVINE RESPIRATORY DISEASE IN FEEDLOT CATTLE

ABSTRACT: Bovine respiratory disease (**BRD**) is one of the most prevalent and economically limiting diseases facing the United States beef industry today. Therefore, the objectives of this study were to (1) characterize the occurrence and prevalence of BRD in feedlot steers utilizing three disease classifications; treatment records (**Trt**), lung lesions present at harvest, and total BRD (treatment or lung lesion or both), (2) to examine the effects of Trt, number of treatments (**NumTrt**), mean lung score, lesions present, and total BRD on economically relevant carcass traits, and(3) to estimate heritabilities for BRD classifications and associated genetic and environmental correlations with economically relevant carcass traits. Data included health and carcass records on 2,870 crossbred steers managed in a commercial feedlot in Southeast Colorado over a two year period. Disease prevalence varied from 45% (n = 698) to 7% (n = 94) in years one and two, respectively. Averaged across the two year period, 27.6% of the steers exhibited clinical signs and were treated for BRD. Lung lesions were collected on 1,226 and 1,261 steers in year one and year two, respectively. Lesions were present in 71% in of steers year one and 47% in year two, and were present in 59% (n=1,461) of the steers over the two year period. Incidence for total BRD, was 76%, 59%, and 64% for years one, two, and overall, respectively. A subset of data (n = 1,260) of animals with

birth information was used to evaluated the effect of age ($P > 0.05$) and BRD classification on carcass traits. Similarly, two models were fitted to the complete data to test the effect of receiving BW as an approximation for age. Evaluation of the models indicated receiving weight to be a significant fixed effect for prediction of carcass merit ($P < 0.001$). Incidence of BRD in the feedlot had a negative impact on both marbling score (**MS**) and subcutaneous backfat thickness (**Fat**) ($P < 0.001$), while accounting for minimal variation ($P > 0.05$) in HCW and LM area after adjusting for fixed contemporary group effects and receiving BW. However, animals that were chronically infected (at least 3 treatments) had reduced HCW (-16.5 ± 4.5 kg) and LM area (4.9 ± 1.25) ($P < 0.05$) compared to steers that were never treated. Lesions present at harvest did not have a significant effect ($P > 0.05$) on any evaluated traits. Animals that were categorized as suffering from BRD by the definition of total BRD had reduced MS (6.1 ± 2.8) and Fat (0.39 ± 0.18) ($P < 0.05$) when compared to healthy steers. Heritability estimates of BRD susceptibility were 0.15 ± 0.06, 0.04 ± 0.03, 0.0 ± 0.0, 0.04 ± 0.06, and 0.07 ± 0.06 for Trt, NumTrt, mean lung score, lesions present, and total BRD, respectively. Genetic correlations were not estimated for mean lung score due to the lack of genetic variability. Genetic correlations of Trt with carcass traits were unfavorable (0.19 ± 0.30) for HCW and LM area (0.03 ± 0.25), and favorable for MS (-0.30 ± 0.21) and Fat (-0.004 ± .26). Genetic correlations for NumTrt were similar at 0.23 ± 0.42, -0.05 ± 0.35, -0.29 ± 0.29, and -0.06 ± 0.35, between HCW, LM area, MS, and Fat, respectively. Estimates of genetic correlations for presence of lesions were zero for all traits. Estimates for total BRD were opposite when compared to Trt, and NumTrt at -0.03 ± 0.4, -0.35 ± 0.36, 0.28 ±0.30, and 0.12 ± 0.35 between HCW, LM area, MS, and Fat, respectively. Results

indicate that with selection genetic improvement can be made over time by utilizing feedlot health records. Genetic correlations between treatments records and carcass traits were in general favorable and would increase profitability when incorporated into selection programs.

Key words: beef cattle, bovine respiratory disease, carcass trait, lung lesions

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1 Introduction and Objectives

1.1 Introduction

Bovine respiratory disease (**BRD**) is the most common and has the largest effect on profitability when compared to other diseases in the United States cattle industry. Bovine respiratory disease is a complex disease that affects all stages of cattle production and cattle at any age. Commonly known as shipping fever in the feedlot segment, the disease accumulates costs due to increased labor and use of pharmaceuticals, decreased production, and increased investment losses associated with mortality.

With increased incidence rates of BRD and associated increases in morbidity and mortality across the United States (Loneragan et al., 2001), it is apparent that the industry has failed as a whole to effectively reduce or eradicate BRD, despite attempts at improvements in the application of pharmaceutical and management practices. As the pathology is further characterized for the causative agents, there is potential for a vaccine to be engineered against most associated microbial and viral agents. Though this may reduce indirect cost associated with production loss, it will also increase pharmaceutical costs on a per animal basis. Consumers of the beef industry have become increasingly aware of animal well being, treatment, and the environmental conditions in which they are raised. The consumer base also expects an increasing amount of meat products to be free from antimicrobial and therapeutic drug residues used in the treatment of infected animals. Concern is also increasing over the potential overuse of antibiotics in animal agriculture, and the implications that may have on the development of antibiotic resistant

organisms in human medicine. These issues warrant research focused on alternative methodologies for the reduction of BRD in the cattle industry.

One of the issues facing disease management is correctly identifying the phenotypes which accurately measure and diagnose the disease and causative agents. Improper classification of diseased animals may lend to an underestimated heritability. Breed differences and heritabilities have been estimated for several species covering a wide range of infectious disease most of which indicate genetic variation within populations. Incidence of BRD may be reduced through selection for individuals with higher resistance and/or tolerance within populations. Previous research has illustrated the success of selection for disease resistant animals in beef, dairy, sheep, poultry and swine industries (Utech et al., 1978; Belt et al., 1995; Bishop et. al., 1996; Mallard et al., 1998; Bishop and Stear, 1999; Wilkie and Mallard,1999, 2000; Heringstad et al., 2000; Zwald et al. 2004a, 2004b). Due to the economic loss associated with the disease, BRD can be considered an economically relevant trait (**ERT**), and therefore methodologies should be investigated for genetic improvement. However, it is imperative that potential genetic antagonisms among BRD and other ERT are known for the successful implementation of any breeding objective that includes the reduction of disease incidence.

1.2 Objective

The objectives of this study were (i) to characterize the occurrence of BRD in a population of feedlot steers through the use of health and lung lesion records, (ii) to evaluate the effect that different classifications of disease have on HCW, LM area, marbling score, and subcutaneous backfat thickness, and (iii) to estimate genetic parameters for BRD based on disease classification and associated genetic relationships with economically relevant carcass traits for the use of genetic improvement for animals with reduced susceptibility to BRD.

2 Review of Pertinent Literature

2.1 An Overview of the Bovine Respiratory Disease Complex

Bovine respiratory disease (**BRD**), commonly known as shipping fever in feedlot cattle and enzootic calf pneumonia in young dairy calves is a vague term used to describe a complex of infectious pneumonia in cattle. Bovine respiratory disease is the most prevalent and costly disease afflicting the cattle industry across the United States today (Yates, 1982; Loneragan, 2001; Callan and Garry, 2002). The generalization of the disease is due to complex interactions between physical, environmental, and epidemiological factors, including both viral and bacterial pathogens (Yates, 1982). The multifaceted nature increases the complexity of disease prevention and management as disease incidence and outbreaks cannot be attributed to a single causative agent. The level of interaction between the physical, environmental, and exposure to pathological agents (viral and bacterial) determines the occurrence and severity of BRD incidence within a given population which then alters the probability of pathogen exposure.

2.2 Causative agents of Bovine Respiratory Disease

Incidence of BRD may occur in animals of any age and at any stage of the production cycle, most commonly affecting young dairy and veal calves, and cattle experiencing production cycle transitioning such as at weaning and/or entrance into the

feedlot. The occurrence of BRD is a synergistic effect of a multitude of causative agents including epidemiological, physical, and environmental agents.

Epidemiological factors affecting incidence of BRD include microbial agent(s), mode of transmission, parasite density, infectious and latent periods, and virulence factors of the various infectious agent(s) (Callan and Garry, 2002). Common bacterial pathogens associated with BRD are *Mannheimia haemolytica* (formerly known as *Pasteurella haemolytica*), *Pasteurella multocida*, and *Haemophilus somnus* (Ellis, 2001; Cusack et al., 2003; Plummer et al., 2004). These bacterial pathogens are ubiquitous to clinically normal cattle populations. The bacteria commonly associated with BRD are considered normal flora within the nasopharynx of some animals, but may not be isolated from all individuals within a given population. Bacterial infection does not occur until the animal has become immunocompromised, allowing opportunistic bacterial pathogens (found in the nasopharynx) to colonize the lower respiratory tract including trachea, bronchi, and lungs. Bacterial infections commonly cause bronchopneumonia resulting in pulmonary lesions.

In most cases, bacterial pneumonia is a secondary infection, frequently prefaced by more common viral infections in the upper respiratory tract, such as bovine herpes virus 1 (**BHV1**); the causative agent of infectious bovine rhinotracheitis (**IBR**), bovine viral diarrhea virus (**BVDV**), bovine respiratory syncytial virus (**BRSV**), and parainfluenza type 3 (**PV-3**) (Ellis, 2001; Callan and Garry, 2002; Cusack et al., 2003; Plummer et al., 2004). These viral pathogens infect the upper respiratory tract, resulting in rhinitis, tracheitis, and bronchitis but have little direct relationship to pulmonary disease (Callan and Garry, 2002). All of the viral pathogens predispose the lungs to

bacterial infections and bronchopneumonia, but BRSV, as the primary agent, has the capability of causing severe lower respiratory tract damage.

Bovine viral diarrhea virus can have a profound affect on BRD incidence within a population. The prevalence of BVDV within the cattle industry has been reported as ranging from 0.3% to 0.4% and 2.6% to 2.5% within chronically ill and dead cattle, respectively (Loneragan et al., 2005; Fulton et al., 2006). Persistently BVDV infected cattle (BVD-PI) can be associated as a main cause of the increased BRD epidemic. Persistently infected cattle occur when a fetus is infected with a noncytopathic strain of BVDV in the first 125 days in utero (Kahrs, 2001). The exact timing of gestational development of immune tolerance is still debated, but exposure must occur before the complete development of a competent fetal immune system. The infected fetus recognizes the virus as "self" during the development of its immune system, and therefore never rids itself of the virus and becomes a persistent shedder of the virus. Therefore BVD-PI cattle are affective in initiating outbreaks of BRD in otherwise healthy cattle within a population (Fulton et al., 2005). Early identification of BVD-PI animals is important in BRD prevention. A survey of randomly selected beef herds in 5 US states revealed 2.7% of the herds had at least 1 BVD-PI calf (Wittum et al., 2001). Persistent infection ultimately results in death of the animal when exposed to a cytopathic infection of BVDV. Wittem et al. (2001) estimated 18.5% of BVD-PI calves die before weaning. However, Larson et al. (2004) estimated that 17 to 50% of BVD-PI cattle could reach breeding age. Similarly Loneragan et al. (2005) estimated that 33% of BVD-PI cattle would survive until slaughter. This increases the risk of exposure of healthy animals by 43% to BVD throughout the production cycle.

Bacterial, viral, and viral-bacterial combination pathogens compromise the respiratory tract defenses, ultimately leading to pulmonary disease. Bovine respiratory disease is ultimately characterized as a summation of physical attributes and environmental factors that allow colonization of common pathogens. Figure 2.1 illustrates some of the multifaceted affects associated with BRD incidence in both the preweaning and postweaning phases of production. Physical preweaning factors attributing to BRD susceptibility center on immune system development (Duff and Galyean, 2007). These factors include neonatal development, nutrition, exposure to BVD-PI cattle (explained above), and farm disease management protocols. Similarly, Callan and Garry (2002) stated that immunological background, age, and stress (both physical and nutritional) played a roll in BRD incidence in preweaned cattle. Postweaning physical factors include stressors of cattle such as shipping and handling, nutrition, and prophylactic prevention treatments (Duff and Galyean, 2007). A culmination of these factors plays a role in the future health and performance of each individual through the production cycle.

Weaning and entrance into the feedlot are events that amplify predisposing causes of BRD. Edwards (1996) divided cattle received into the feedlot into high and low risk categories based on physical and environmental backgrounds. High risk animals included freshly weaned calves, cattle that had been transported for at least 20 hours, cattle that had been assembled at auction markets (comingled from various sources), and cattle that appeared to be highly stressed when received at the feedlot. Low risk category included yearling cattle from one source, cattle that arrived from a backgrounding operation where they had been in a feedlot-like environment, and low stressed calves that had been

weaned. It is commonly accepted within the industry that lighter cattle pose a greater health risk when entering the feedlot than do heavy cattle (Lekeux, 1995; Edwards, 1996; McAllister et al., 2008).

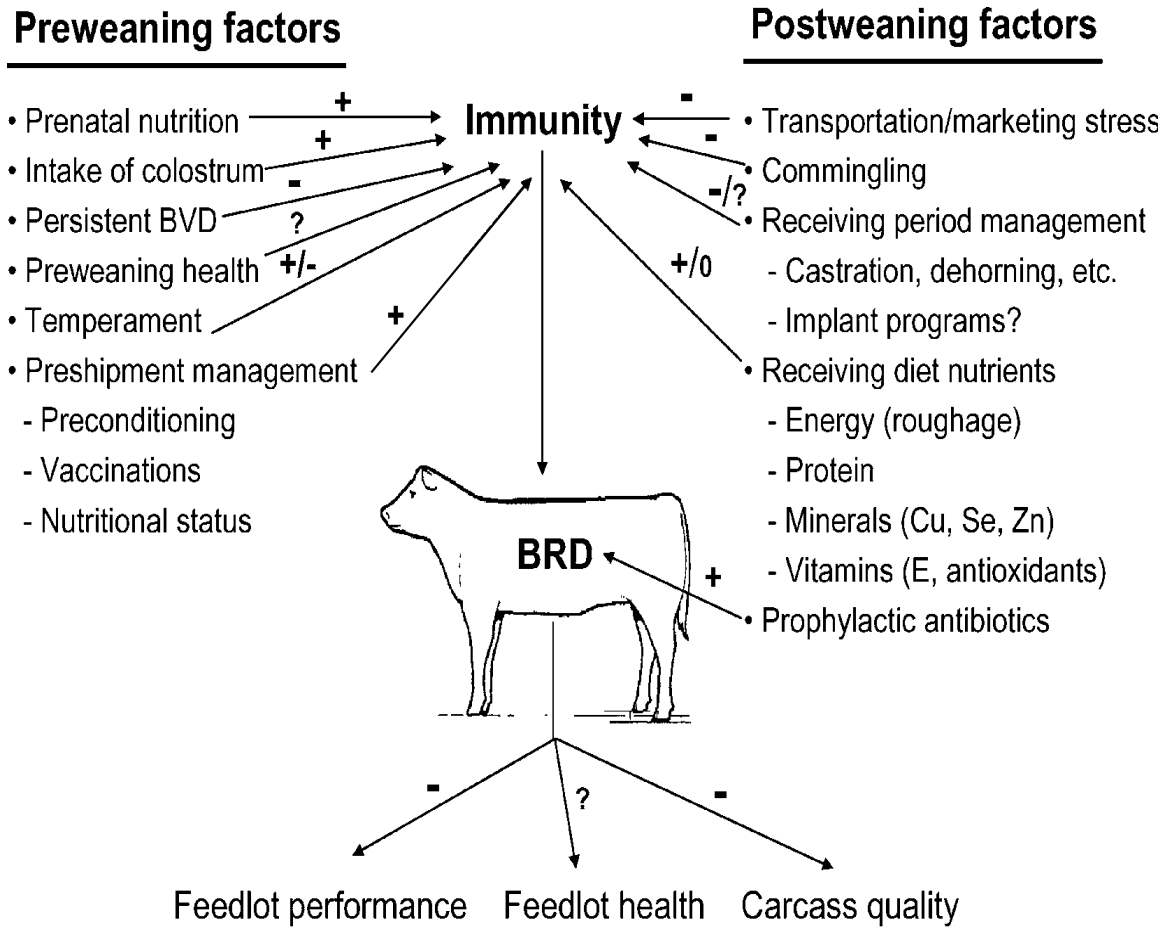

Figure 2.1. Pre and postweaning factors affecting bovine respiratory disease (BRD) in beef cattle and the resulting outcomes of the disease.
+ = decreased incidence or consequence; − = increased incidence or consequence; ? = effects not fully understood based on the available data. BVD = bovine viral diarrhea virus. Adapted from Duff and Galyean (2007).

Cattle received into the feedlot weighing less than 450 kg are at higher risk of contracting BRD than those weighting more than 450 kg (Edwards, 1996). Similarly, McAllister et

al. (2008) reported that cattle received into the feedlot at lower weights were more likely to display clinical signs of BRD early in the feeding period, associated with receiving stress, than those arriving at higher weights. Lekeux (1995) attributed BRD incidence in lighter calves to underdeveloped respiratory systems related to younger ages.

Commingling of cattle from multiple sources (sale barn cattle), or cattle with varying immunological backgrounds have also proven to increase BRD rates. O'Connor et al., (2001) found a 120% increase in chronic disease in commingled cattle compared to feedlot pens with cattle from a single source. Similarly Step et al. (2008) found that preconditioned calves were healthier (had lower morbidity rates) and performed better than multiple-source market calves. Stress from travel and processing procedures have also been shown to increase incidence of disease. Brigham et al. (2008) found that processing stress, measured by length of time spent in a processing chute, and processing order (time spent away from feed and water) increased an animal's likelihood of displaying clinical signs of BRD and consequently being treated.

There are also a vast number of environmental factors that predispose both preweaning and postweaning cattle to BRD. These environmental factors include climate, ambient temperature, dust particles, and stocking densities (exposure to agents) (Callan and Garry, 2002; Duff and Galyean, 2007). In the United States, there is a seasonal influx of weaned (ages 5 to 8 months) and backgrounded cattle (ages 8 to 12 months) entering the feedlot. This influx of cattle entering the feedlot is generally seen during the fall and early winter, a time associated with rapid weather changes and temperature extremes. Speidel et al. (2008) found dramatic changes in temperature extremes increased the probability of animals being identified with clinical BRD during the early feeding period.

2.3 Clinical and Subclinical Symptoms of Bovine Respiratory Disease

Clinical and subclinical BRD is a product of immunosuppressive stressors allowing colonization of the respiratory tract by opportunistic pathogens commonly encountered as normal flora in the upper respiratory tract. These physical, environmental, and epidemiological stressors are exacerbated during weaning and feedlot production stages, leading to increased incidence of clinical BRD, as discussed above. Acceptance on an industry wide basis that a variable but high percentage of animals will become infected with BRD makes accurate diagnosis essential. The majority of cases will be identified and treated before the first 27 days of the feeding period (Jensen et al., 1976; Buhman et al., 2000; Radostits et al., 2000). The most common clinical symptoms associated with BRD are:

1. Respiratory distress (labored breathing)
2. Cough
3. Nasal and/or ocular discharge
4. Depression
5. Anorexia
6. Fever (increased rectal temperature)
7. Death

Clinical symptoms may vary in the way they present from individual to individual. The variation in which the disease may present makes diagnosis of BRD difficult. Duff and Galyean (2007) recommend that observation of any combination of the clinical

symptoms listed above, including a rectal temperature of over 40 degrees C° (104 F°) is indicative of BRD. Given the subjective nature of the diagnosis, accurate identification of infected animals is not always possible. The animal's symptoms may present subclinically and therefore go undetected by feedlot personal. It is not well known why, when infected, some individuals within populations present clinical symptoms and others present subclinically. Noffsinger and Locatelli (2004) attributed subclinical expression of symptoms to a predator/prey response. They proposed that animals would suppress their clinical symptoms as to not be perceived as weak in the presence of humans (predators), and therefore symptoms would go undetected by feedlot personnel.

Detection of subclinical individuals is impossible and therefore true disease prevalence is often underestimated as well as the associated affects of the disease on production and profitability. Several studies have investigated the use of pulmonary lesions at harvest to accurately estimate "true" prevalence of BRD within populations. Lesions in the cranial ventral lung lobe have been found to be adequate in identifying subclinical BRD (Bryant et al., 1999). Epperson (2003) further supported the findings of Bryant et al. suggesting that lung scoring of only the right cranial and right middle lobes performed at commercial processing speeds would diagnose 86.1% of lung lesions. Pulmonary lesions at harvest have been reported to increase accurate estimation of true disease prevalence, and better predict the reduced performance during the finishing period when compared to BRD treatment records alone (Wittum et al., 1996; Bryant et al., 1999; Thompson et al., 2006). Table 2.1 shows treatment rates and percentage of lung lesions by treated and non-treated animals from a number of studies. Incidence of BRD, defined as being identified for treatment, ranged from 8.17-35%. Interestingly 42 to 70%

of each of the populations presented pulmonary lesions at harvest. All studies showed a large proportion (38.8-60.6%) of the untreated animals presented with pulmonary lesions at harvest indicating unidentified BRD challenges. These are relatively large proportions of animals that are going untreated and/or displaying subclinical symptoms. Schneider et al., 2009 attributed the 60.6% of animals presenting with lung lesions from those not treated for clinical BRD to (i) feedlot personal simply missing a large number of cattle that had suffered from BRD, (ii) many animals suffered from subclinical disease, (iii) some observed lesions were from instances of BRD exposure before arrival in the feedlot, or (iv) a combination of the above three events. On the other hand all studies also demonstrated that there was a proportion (9.9-63%) of the animals that were treated for BRD during the feeding period and presented no pulmonary lesions at harvest. These animals could be considered to be (i) misdiagnosed or (ii) successfully treated thereby preventing any lung damage from the infection. All studies indicated an increase in disease prevalence when including pulmonary lesion scores at harvest in addition to treatment records, compared to using treatment records alone. Similarly the inclusion of pulmonary lesion scores improved estimation of production loss and increased days on feed better than treatment records alone. This illustrates the difficulty feedlot personal experience in accurately detecting BRD incidence. Identification of pulmonary lesions at harvest does little to benefit and improve treatment of infected animals while in the finishing period. However, there is potential to use information from both phenotypes (treatment records and lung lesion scores) in selection of breeding animals who are less susceptible to BRD.

Table 2.1. Comparison of lung lesions present at harvest and treatment records for BRD

n	Treated[1]	Not Treated[1]	Treated No Lesion[2]	Not Treated With Lesions[2]	Source
469	35.0% (164)	65.0% (305)	22%	68.0%	Wittum et al., 1996
439	17.0% (75)	83% (364)	63.0%	42.0%	Bryant et al., 1999
2,036	22.6% (460)	77.4% (1,576)	9.9%	38.4%	Thompson et al., 2006
1,665	8.17% (136)	91.83% (1,529)	26.0%	60.6%	Schneider et al., 2009

[1] Percentage of total (n)
[2] Percentage of the number from each category (Treated/Not Treated)

2.4 Prevention and Management Practices

Methods currently implemented to control disease include sanitation, quarantine, culling, vaccination and therapeutic medications. Over the past several years there has been considerable effort put forth by both veterinarians and researchers to better understand and prevent BRD. Most of the research has been directed towards the areas of pharmaceuticals and management practices, as these are the easiest methods by which prevention is implemented currently (Bagley, 1997).

Management practices that reduce pathogen introduction, exposure, and transmission are important steps in managing BRD (Snowder et al., 2006b). Vaccines can be effective for reducing not only susceptibility but also for reducing shedding of infectious BRD agents to other calves (Frank et al., 2002, 2003). Table 2.2 illustrates the therapeutic products feedlots in the United States use to treat cattle for an initial course of BRD to minimize disease incidence and shedding. As the cattle industry's consumers become more health aware, there is increasing skepticism about antimicrobials,

pharmaceutical, and hormone residues in meat products and their associated animal and consumer health. This distrust necessitates development of alternative management practices to decrease the use of antibiotic therapies in the prevention and treatment of BRD.

Table 2.2. Percent of Feedlots by product typically used to treat cattle for an initial course of BRD, by feedlot capacity

	Feedlot Capacity (Number of Head)		
Therapeutic Product	1,000-7,999	≥ 8,000	All Feedlots
Injectable antibiotic	99.8	100	99.8
Oral antibiotic	31.1	16.5	27
Vitamin C injection	6.1	16	8.9
Vitamin B injection	31.8	30.3	31.4
Respiratory Vaccine	31.5	64.1	40.6
Corticosteroid	20.4	27.1	22.3
Non-steroidal anti-inflammatory drug	37.7	47.6	40.5
Antihistamine	31.6	37.5	33.3
Anthelmintic (dewormer)	8.7	7.1	8.3
Probiotic paste	31.9	23.1	29.5
Oral electrolytes, fluids, drenches	20.2	33.4	23.9
Other	1.3	1.8	1.5

Adapted from USDA APHIS (2001)

Preconditioning, used to reduce morbidity and mortality rates of feedlot cattle, is not a new practice in the beef industry, however an industry wide adoption of the practice has been slow. Preconditioning of calves has been described as a comprehensive

management system designed to immunize calves against common BRD viral agents and minimize stress responses during the transition processes from weaning to the feeding period (Cole, 1985; Speer, 2001; Dhuyvetter et al., 2005; Duff and Galyean, 2007. A review of various preconditioning protocols reported increases in gain both on-farm and within the first 45 days of the feedlot period; and reductions in both morbidity and mortality rates by 23% and 0.7%, respectively, when compared to non-preconditioned cattle from the same source (Cole, 1985). Similarly Step et al., (2008) found preconditioned calves weaned on the ranch for 45 days before transporting to a receiving facility resulted in improved health and performance during the subsequent receiving and feeding period compared with weaning and transporting calves immediately, or purchasing calves from market. The American Academy of Bovine Practitioners (1968) defines a preconditioning protocol as one containing the following criterion:

1. Calves weaned at least 3 weeks before sale.
2. Calves trained to eat from a feed bunk and to drink from a trough.
3. Calves treated for parasites.
4. Calves vaccinated for blackleg, malignant edema, parainfluenza-3 virus (PI-3), IBR, BVDV, *Pasteurella*, and *Haemophilus somnus*.
5. Calves castrated and/or dehorned
6. Calves identified with an individual ear tag

Preconditioning serves as a "preschool" scenario preparing calves for transitional periods in an effort to build up immunity and reduce both mental and physical stressors upon entrance into the feedlot.

To increase industry acceptance of preconditioning, economic incentives must be shown. Dhuyvetter et al. (2005) suggested based on a 45 day postweaning preconditioning program cow/calf producers can realize a $14.00 increase per calf in returns compared with the sale of calves at weaning that are not preconditioned. At this rate of return, feedlot producers also can afford to pay premiums for preconditioned calves. Others have estimated the premiums paid to cow/calf producers as ranging anywhere between $0.041 and $0.121 per kg (McKinnon and Greiner, 2002; Avent et al., 2004). Despite the increased benefits seen from preconditioning and on farm vaccination programs through increased health of the cattle, only 28.4% cow/calf producers vaccinate against causative agents (USDA APHIS, 1997; USDA NAHMS, 2000a). Despite the increased incentives that could be obtained by producers, Purdy et al. (1987) showed that the percentage of cow/calf producers in seven Southeastern states who vaccinate for a pathogen related to BRD was only between 10.3 to 27.6%.

The finishing segment stands to gain from encouraging cattle suppliers to implement preconditioning systems as well. Studies have shown that there is a $46.00 to $60.00 increase per head in return at harvest from calves that have been preconditioned into the feedlot compared to those that were not (Cravey, 1996; Roeber and Umberger,2002). Faber et al. (1999) found that calves that were not weaned before entrance into the feedlot had a 3.4 greater chance of being affected with BRD than those that were weaned more than 30 days before entrance. Similarly Roeber and Umberger (2002) found a 41.6% reduction in BRD incidence between the two groups of preconditioned and non-preconditioned cattle.

Once an animal has entered the feedlot, many commercial feedlots will implement different preventative protocols based on risk assessment of the cattle received. However pre-arrival processing information is rarely known on newly arrived cattle. Approximately 67.6% of feedlots never or only "sometimes" know the pre-arrival information (USDA NAHMS, 2000b). Therefore, it is common practice that these producers vaccinate all cattle against common BRD viral agents (IBR, PI-3, BVDV, and BRSV) upon arrival due to the lack of background information. Most feedlots operate under the "pull and treat" method based upon pen riders' observation of clinical symptoms. Treatment varies based on a variety of commercially available pharmaceutical drugs. Proper antimicrobial treatment may be determined based on clinical response, necropsy, field trials, and cost (Smith, 1992). Ultimately viral vaccines and other antimicrobials are used to reduce incidence and severity of a disease outbreak, but are limited in terms of their ability to prevent and eradicate BRD from entire populations. Currently there are several BRD vaccines available (Perino, 1997), none of which cover all causative pathogens.

When cattle are high risk or in the cases of severe outbreaks within a population, the management practice of metaphylaxis (mass medication) is commonly implemented. Mass medication of infected animals has been shown to be highly successful in reducing morbidity rates from 60% to as low as 7% in control and mass medicated groups (Lofgreen, 1983; Morck et al., 1993; McCoy et al., 1994; Merrill et al., 1994; Galyean et al., 1995). Galyean et al. (1995) also showed that simply mass medicating on rectal temperature alone reduced the number of animals treated (100% to 42%). This procedure minimized unneeded treatments and reduced pharmaceutical costs while decreasing total

morbidity. There are large drawbacks to metaphylaxis treatment of large groups of cattle. There are increased pharmaceutical costs and a risk of developing antibiotic resistant organisms through treatment of healthy animals. Few antibiotics have been developed in the past 30 years (Detilleux, 2001), therefore extreme caution and judgment is recommended before implementing a mass medication protocol and suggest that this treatment be limited to only extremely high risk animals.

Despite the current understanding of BRD, the ongoing prevalence in cattle populations illustrates that the efforts put forth for prevention and treatment of the disease have failed on an industry wide basis. Therefore, future research seeking alternative methods to reduce BRD incidence in addition to, or possibly in place of, historical pharmaceutical therapies and management practices is warranted.

2.5 Economic Impacts of Bovine Respiratory Disease

The economic impacts of any disease are vast, and range from treatment, pharmaceutical expense, increased handling and labor, indirect costs associated with reduced animal productivity and/or lower value for chronically infected individuals, increased time on feed, and cost associated with mortality (especially on individuals late in the feeding period). BRD is unique in that economic losses can occur in all expense categories, when compared to other diseases encountered in the feedlot such as pinkeye or lameness which generally are not associated with any mortality loss. The success and profit of the beef industry are greatly important to the economy, as this industry is the single largest enterprise in United States agriculture at $188.4 billion (Otto and Lawrence, 2001).

Bovine respiratory disease causes approximately 75% of the morbidity and over 50% of the mortality in feedlots (Edwards, 1996; Smith, 1998). Edwards (1996) and Vogel and Parrot (1994) analyzed feedlot records and divided the causes of morbidity and mortality of feedlot cattle (those in sick pens) by the body system affected. Results from these studies are presented in Table 2.3. Edwards (1996) found over 67% of the morbidity rates occurring within the feedlot were attributed to BRD. Similarly, and as would be expected with increased morbidity rates attributed to BRD, mortality rates were higher in those animals infected with BRD than those suffering from digestive or miscellaneous complications. USDA APHIS (2001) reported incidence rates of clinical BRD to be 14.4%, from a study of feedlots with greater than 1,000 head capacity over 12 states.

Table 2.3. Incidence of morbidity and mortality in large feedlots

		Percentage of total disorders			
Breed type	Incidence rate	Respiratory	Digestive	Miscellaneous	Source
Beef[1]	5-11%	67-82%	3-7%	14-28%	Edwards (1996)
Beef[2]	0.57-7.07%	46-67%	14-36%	16-22%	Edwards (1996)
Beef[2]	0.27% /Month	44.10%	25.90%	28.60%	Voegel and Parrot (1994)
Holstein[2]	0.42% /Month	35.50%	42.40%	15.40%	Voegel and Parrot (1994)

[1]Measurements of morbidity
[2]Measurements of mortality

The majority of management procedures for prevention and treatment of BRD involve pharmaceutical therapies as discussed above in section 2.4 *Prevention and Management Practices*. Treatment and prevention for BRD have been estimated to cost the beef industry more than $700 million annually (Griffin, 1997; National Agricultural Statistics Service, 2006). Results from the 1992-1995 Texas A&M Ranch to Rail study

(Anonymous, 1993-1995) illustrated that the expense of treatment could range from $20.76 to 37.90 per animal treated. In a nationwide study performed by USDA APHIS (2001), those feedlots that used the most common combinations of two product types, reported costs to treat one animal at $8.80 (oral and injectable antimicrobials); $12.36 (vaccine and injectable antimicrobial); $11.73 (NSAID and injectable antimicrobial); and $11.64 (probiotic paste and injectable antimicrobial). The study also found that it was more expensive to treat one animal for BRD than one for acute interstitial pneumonia (AIP), digestive disorders, buller syndrome, lameness, and central nervous system disease (CNS). Costs also increase for cattle that are chronically ill and require multiple treatments. Fulton et al. (2002) found calves that were not treated to return $40.64, $58.35, and $291.93 more when compared to calves treated 1, 2, and 3+ times, respectively. These estimates include costs for pharmaceuticals, but do not take into consideration time, labor, and production loss. McNeill et al. (1996) estimated a $92.26 greater profit from non-sick animals than from sick. Only $31.00 of the difference in profits were due to pharmaceutical costs, with the remainder being due to reduced efficiency, lowered gain, and reduced sale values of sick cattle.

The effect BRD has on performance traits in the feedlot has an overall negative impact on profitability. It has long been accepted that calves treated for BRD have lowered performance when compared to calves not treated for BRD. Literature shows a large difference in ADG in the early feeding periods between treated and non-treated cattle; however, studies have illustrated little difference (0.07 kg) in ADG in the total feeding period between the two groups (Bateman et al., 1990; Gardner et al., 1999; Schneider et al., 2009). The small difference in ADG during the entire feeding period

between treated and nontreated animals can be attributed to some compensatory gain attained by the infected animals after recovery. However, Schneider et al. (2009) reported a difference in animal value due to ADG of $15.76, $22.09, and $46.70 when comparing cattle that were never treated for BRD with cattle that were treated 1, 2, and 3+ times, respectively.

The beef industry has increased the percentage of cattle that are being marketed on a carcass merit basis through various grid and branded product systems. Therefore, the economic cost of BRD on reduced carcass performance has increased in significance over recent years. The increased concentration of animals from varying environmental and immunological backgrounds increases the probability of pathogen exposure. When cattle are sold on a live-weight basis, economic losses are confined to death loss, treatment cost, decreased fed efficiency and decreased live weight. When cattle are sold on a carcass merit basis, disease has the potential to affect not only carcass weight, but also the quantity, location, and ratios of muscle, fat, and water (Larson, 2005). Bovine respiratory disease has been shown in several studies to influence carcass traits such as carcass weight, marbling, and subcutaneous fat cover (McNeill et al., 1996; Gardner et al., 1999; Stovall et al., 2000; Roeber et al., 2001; Babcock et al., 2009; Schneider et al., 2009). These studies showed that cattle suffering from clinical BRD tended to have lower hot carcass weight, less external and internal fat, and less desirable yield grades than did steers without clinical signs of BRD. Hot carcass weight, dressing percentage, adjusted fat thickness, and yield grade also suffered as number of treatments for an animal increased (Gardner et al., 1999; Roeber et al., 2001; Schneider et al., 2009). Though incidence of BRD has a negative effect on most carcass traits, both Gardner et al. (1999)

and Roeber et al. (2001) found BRD to have no impact on shear-force values, tenderness ratings, or juiciness ratings. Schneider et al. (2009) found decreased economic value attributable to differences in quality grade when compared with cattle that were never treated. These reductions were reported at $7.48, $9.58, and $7.70 for 1, 2, and 3+ treatments, respectively. Though BRD has been found to influence carcass composition there has been no direct pathway linking disease to changes in carcass traits. Larson (2005) provided three theories for potential pathways between disease and changes in carcass characteristics; (i) a change in metabolic signals, such as cytokines and cortisol, could have an effect on carcass composition through modification of hypothalamic secretions of thyrotropin releasing hormone, by inhibition of insulin-like growth factor I and insulin action on muscle and fat tissues, and by direct protein catabolism and lipolysis; (ii) disease-induced anorexia causing a decrease in serum insulin-like growth factor I and an increase in serum growth hormone, which induces a change in the partitioning of nutrients for tissue deposition; and (iii) there is an indirect (and reversible) effect of anorexia whereby sick cattle are on feed for fewer effective days than pen-mates that do not become sick assuming the entire pen is harvested at the same time. Other pathogen or immune-mediated responses to disease, as well as interactions among hormones and cytokines, may influence nutrient partitioning and body composition but have yet to be described.

The greatest cost associated with BRD is mortality, particularly late in the feeding period as opportunity cost increases and there is a 100% loss of salvage value. Loneragan et al. (2001) estimated that the rate of mortality due to BRD is on the rise, with BRD representing 57.1% of all deaths with rates increasing from 1994 to 1999 by 38%.

Mortality due to BRD cost the industry $692,430,000 in 2005. Economic losses attributed to BRD are considerably greater than losses associated with digestive problems; which is the second greatest factor for death loss in the U.S. beef industry at $367,441,000 (USDA NASS, 2006). USDA NASS, (2006) also reported BRD to be the cause of death for 37.7% of bulls, cows, and heifers.

2.6 Selecting for Disease Resistance

Genetic improvement of health traits has the potential to reduce costs associated with isolation, treatment, decreased production, and culling of infected animals. It also has the allure of improving animal welfare and easing consumer concern of antibiotic and therapeutic drug residues in meat products. The successfulness of a genetic improvement program depends on the genetic variation within a given population (heritability), and the amount of selection pressure placed on the trait of interest. Selective variation for resistance to specific pathogens has been found to be present in some, if not all, animal populations (Nicholas, 1987).

One of the biggest questions facing the livestock industry relative to selection for health traits is whether selection emphasis is being placed on increased (i) resistance, the ability of an animal to resist infection or moderate the bacterial, viral, and/or parasitic lifecycle, (ii) tolerance, the animals ability to withstand the pathogenic effects of infection, or (iii) decreased susceptibility; i.e., culling animals that are at a higher risk of infection within the population. The answer depends on whether we want to manage disease incidence and transmission or simply minimize the effect infection has on production traits. Moreover, genetic improvement and the success of selection for disease

resistance is dependent on correctly identifying the phenotype for disease resistance (Snowder, 2006a). This is generally difficult due to the binary nature of disease incidence of infected versus not. However, the severity of disease is more problematic if used to identify appropriate phenotypes. Proper identification of phenotypes can be difficult when differentiating between clinical versus subclinical symptoms and primary versus secondary infections especially when incidence is low within the population. There is also the issue of whether all animals were sufficiently challenged, and therefore it is a faulty assumption that all healthy animals are disease resistant. There are alternative methods to accurately select for disease resistance through both direct and indirect selection. Direct selection would include: (i) observing for diagnosis of disease or treatment records (ii) challenge all animals equally, which may not be ethical and/or cost effective, and (iii) challenge the relatives of breeding stock. Indirect selection may consist of collecting indicator phenotypes such as: (i) pathogen-related by-products and (ii) measuring biological and/or immunological response of the host (Rothchild, 1998; Snowder, 2006a) .

Genetic improvement of disease resistance has been implemented over multiple diseases and in several species, i.e., cattle ticks (Utech et al., 1978); *E. coli* strains in pigs (Edfors-Lilja and Wallgren, 2000), gastro-intestinal parasites in lambs (Bishop et. al., 1996; Bishop and Stear, 1999), Marek's disease (Cole, 1968; Steadham et al., 1987; Pinard et al., 1993), nematode infection (Bisset and Morris, 1996), scrapie in sheep (Belt et al., 1995), and mastitis (Heringstad et al., 2000). Zwald et al. (2004a, 2004b) demonstrated that a multiple-trait analysis of farmer-recorded disease incidence would

improve health traits for cystic ovaries, displaced abomasums, ketosis, lameness, mastitis, and metritis.

Genetic improvement of enhanced immune responsiveness has proven successful in poultry, sheep, and swine (Mallard et al., 1998; Wilkie and Mallard,1999, 2000). Selection for improved immune response may have great genetic potential due to the large biological component which is controlled by many genes (Glass, 2004). Saltman (1992) reported that each animal has its own inherent (genetically controlled) strengths and weaknesses. Coupled with these genetic factors are the developmental events that occur prior to birth (congenital effects). Thus, there is a great deal of variation among calves in their ability to mount an immune response as early as the day of birth, indicating the possibility of large genetic diversity within populations. However, the energy required to mount a sufficient immune response and the corresponding reduced production needs to be weighed against the benefit of decreased disease (Bishop and Stear, 2003).

If selection proves successful, potential genetic antagonisms between traits of interests must also be considered. One concern with selection for improved disease resistance is potential antagonisms with other diseases and production traits. McEwan et al. (1992) and Raadsma et al. (1997) both studied relationships between disease resistance to several parasite and infectious diseases in sheep. Both studies illustrated that genetic relationships were nonexistent and/or sometimes favorable between diseases when selecting for resistance for a single parasite or infection. Similarly Zwald et al. (2004b) found favorable genetic relationships between disease resistance for multiple diseases in dairy cattle. Genetic improvement of immune response has also shown to

positively improve performance traits of sheep (Bishop et al., 1996) and pigs (Mallard et al., 1998; Wilkie and Mallard, 2000).

Evidence of genetic control for disease resistance is evident in breed differences for various species, and these differences illustrate genetic diversity between populations, and therefore potential genetic variation within populations. Breed differences have also been documented for keratoconjunctivitis (pinkeye) in cattle (Snowder et al., 2005a), *Bordetella bronchiseptica* infection in swine (Rothschild et al., 1984) and immune response in chickens (Zekarias et al., 2002). Generally genetic differences between breeds are used to enhance performance through crossbreeding and heterosis.

In populations that have been challenged by BRD causative agents there are inevitably individuals that are more tolerant, less susceptible, and/or more resistant to disease. The relatively few heritability estimates illustrate that there is genetic variability within the population and therefore selection to improve resistance is possible. Heritability estimates for susceptibility to BRD have been found to be lowly heritable on the observed scale ranging from 0.06 to 0.22 (Muggli-Cocket et al., 1992; Snowder et al., 2005b, 2006b, 2007; and Schneider et. al., 2008).

Table 2.4. Literature estimates of direct heritabilities on the observed scale for pre and postweaning incidence of BRD

	h^2_d[1]		
Breed	Preweaning	Postweaning	Source
Multi-Breed	0.10 ± 0.02	0.06 ± 0.07	Muggli-Cocket et al. (1992)
Multi-Breed	0.22 ± 0.01	-	Snowder et al. (2005b)
Multi-Breed	-	0.08 ± 0.01	Snowder et al. (2006b)
Multi-breed	-	0.08 ± 0.01	Snowder et al. (2007)
Angus Cross	0.12 ± 0.06	0.07 ± 0.04	Schneider et al. (2008)[2]

[1] h^2_d = direct heritability estimate
[2] heritability estimates are on the underlying scale

Heritability estimates for previous research based on breed, preweaning and postweaning incidence are shown in Table 2.4. The lower heritability estimates on the observed scale are not surprising because fitness traits have generally been considered to be lowly heritable (Falconer, 1981). Snowder (2005b) also illustrated that there was potential to identify animals with a high probability of being disease resistant in years of high incidence, but in years of low incidence the accuracy of identification was diminished. This can be explained by the binary nature of disease incidence and as a byproduct of analyzing the trait on the observed scale which relies on the proportion of infected animals present. The low estimates indicate that with increased selection pressure genetic improvement can be made to reduce susceptibility and possibly increase the tolerance of cattle to BRD. However, when the heritability estimate of 0.22 ± 0.01 (Snowder et al., 2005b) was converted to the underlying continuous scale, heritability of BRD resistance in preweaned calves was high at 0.48. This estimate would suggest that

large genetic gains could be made through the use of selection tools if this is indeed the appropriate heritability.

Schneider et al. (2008) reported heritability estimates for number of BRD treatments during both preweaning and feedlot phases to be 0.05 ± 0.04 and 0.08 ± 0.05, respectively. These heritability estimates for number of treatments may indicate genetic variation for resistance and immune response. Though clearly there is still a large environmental component associated with recovery, which may include timely identification and antibiotic treatment of sick animals, and vaccine efficacy.

Genetic correlations between direct genetic and maternal genetic effects for incidence of BRD have been estimated to be high and negative (-0.71 to -0.98) (Snowder et al., 2005b). These large and significant correlations indicate that dams that are superior in resisting BRD infection produce calves that are more likely to be infected with BRD. Indicate that superior dams are supplying passive immunity through milk (Ellis, 2001). However, as passive immunity decays, those individuals from superior dams may be at greater risk of infection due to antagonisms between vaccine efficacy and passive immunity. Calves from younger dams experience higher preweaning BRD but lower postweaning incidence than calves from older dams, possibility due to lower colostral antibody concentration and therefore less passive immunity which gives higher vaccination response during the feedlot phase (Muggli-Cockett et al., 1992). There is evidence that neonatal calves that have been deprived of colostrum are capable of expressing a favorable immune response to a *Pasteurella haemolytica* vaccine without passive immune interference (Hodgins and Shewen, 2000)

Breed differences for resistance to BRD further illustrate the genetic component contributing to BRD incidence within populations (Muggli-Cockett et al., 1992; Snowder et al., 2005b, 2006). Muggli-Cockett et al. (1992) found Gelbvieh, Hereford, Limousin, and Simmental calves to have the lowest incidence of BRD during preweaning phase of production. There was a significant "location" effect, prohibiting direct comparisons of all breed types. They then found significant breed differences in the postweaning period, where direct comparisons could be made over the eleven purebred and three composite breed types. They found purebred Angus, Gelbvieh, and Limousin cattle to have the lowest incidence of BRD in the feedlot while Hereford and Pinzgauer cattle were found to have the highest incidence of BRD. Similarly Snowder et al., (2005b) estimated breed differences during the preweaning phase between British and Continental breedtypes. They estimated favorable solutions for Longhorn, Red Poll, Salers, and Shorthorn. These breeds did not differ for incidence of BRD. Friesian calves were significantly more likely to have BRD infection compared with calves from most breeds. Angus and Hereford calves did not have higher incidences of BRD than other breeds. Snowder et al., (2005b) also estimated dam breed differences and found few differences among breeds. However, he did estimate a negative difference in the solutions for calf and dam breed effects. Suggesting that generally calves produced by dams that had a low estimate for BRD incidence would themselves suffer from a higher incidence of BRD and vice versa.

Breed differences would also indicate that genetic improvement to reduce incidence of BRD could be managed through crossbreeding systems, however there has been conflicting results with Muggli-Cockett et al. (1992) reporting favorable heterotic effects, and Snowder et al. (2005b) reporting negligible effects due to heterosis.

However, it has been greatly accepted that crossbred cattle are healthier than their straightbred counterparts.

The development and implementation of any successful breeding plan must take into consideration potential genetic antagonisms between BRD and other economically relevant traits (**ERT**). Muggli-Cocket et al. (1992) reported positive genetic correlations between preweaning BRD occurrence and birth weight (r_g = 0.25 ± 0.10) and postweaning BRD occurrence and birth weight (r_g = 0.50 ± 0.15). This correlation could be attributed to the stress that heavier calves experience at birth (dystocia) and subsequent failure in immune responses due to passive transfer. Schneider et al. (2008) reported conflicting results with no direct genetic correlation between preweaning BRD incidence and both birth and weaning weight. Though Gardner et al. (1999) and Roeber et al. (2001) found no negative phenotypic associations between shear force and treatment for BRD, Snowder et al. (2007) found that selection for resistance to BRD may have an undesirable correlated effect on shear force (genetic correlation of 0.20 ± 0.16) and a reduction in the percentage of bone weight (genetic correlation of -0.42 ± 0.14). Similarly, Schneider et al., (2008) found genetic correlations between BRD incidence in feedlot cattle and performance traits to be: -0.90 ± 0.20 for warm-up (4-6 weeks) ADG, 0.14 ± 0.25 for on-test ADG, -0.35 ± 0.22 for overall ADG, -0.43 ± 0.21 for final weight, 0.00 ± 0.23 for hot carcass weight, 0.02 ± 0.23 for LM area, -0.03 ± 0.26 for fat, and -0.42 ± 0.21 for marbling score. Though genetic correlations for carcass traits were negative, they were also insignificant with high standard errors. The low and non-significant estimates of genetic correlations between BRD incidence and both production and carcass traits suggest that selection to reduce incidence of BRD in the feedlot would

have minimal effect on growth, carcass, and palatability traits. Inclusion of health traits in selection indices would greatly improve selection of health, performance, and carcass traits of any production system.

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3 Materials and Methods

This chapter describes the source of cattle and animal handling practices that were implemented for the collection of relevant health and carcass data. Specific statistical analysis is presented in subsequent chapters.

3.1 Population

All experimental procedures were reviewed and accepted by the Colorado State University Animal Care and Use Committee (07-230A-01).

Crossbred steer calves were obtained from a single ranch source in Western Nebraska originating from one of three units (**UNIT**) in November of 2007 and 2008 (n = 1,551 and 1,319), respectively. Number of calves obtained from each UNIT is presented in Table 3.1.

Table 3.1. Numbers of calves received from individual ranch units.

	UNIT			
Year	A	B	C	Total No.
2007	719	612	220	1,551
2008	562	552	205	1,319
Total No.	1,281	1,164	425	2,870

3.1.1 Ranch data:

Birth date and weaning information was available on 1,431 calves over the two years. Calves were born between February through May with a mean weaning age of 135 d and a mean weaning weight of 157.96 Kg (Table 3.2). Calves were weaned and backgrounded for an average of 83d before delivery (70 d and 96 d for year one and year two, respectively) to the commercial feedlot. The average receiving weight at the feedlot was 223.9 Kg and 219.1 Kg for year one and two, respectively.

Table 3.2. Summary of calf ages, weights, and backgrounding on ranch and at receiving.

Trait	No.	MEAN	SD	MIN[1]	MAX[1]
Weaning Age	1,376	135	21.27	72	191
2007	791	134	22.83	91	191
2008	585	137	18.86	72	173
Weaning Weight	1,395	158.0	26.48	81.7	240.4
2007	802	161.1	25.49	90.7	236.8
2008	593	153.7	27.21	81.7	240.4
Background[2]	1,394	83	22.27	51	113
2007	802	78	22.89	51	110
2008	593	89	19.57	56	113
Receiving Weight	2,864	221.7	24.34	150.6	335.7
2007	1,551	223.9	23.46	156.0	324.8
2008	1,313	219.1	25.11	150.6	335.7

[1]Min = Minimum value; Max = Maximum Value
[2]Background = length in days

3.1.2 Pedigree information

Calves in the study were a result of multi-sire breeding pastures. Sire identification was performed by DNA sampling of both sire and progeny through a commercial lab. Eighty one percent of calves were successfully sire identified (n = 2,331). A historical pedigree was obtained from the ranch of origin. A five generation sire pedigree (n = 3,255) was constructed for the purpose of variance component estimation. The pedigree consisted of 386 sires; percentage of sires with two or more and five or more progeny were 80 and 51%, respectively.

3.2 Processing

Cattle were shipped from Western Nebraska to a commercial feedlot in Southeastern Colorado. Cattle were in transit approximately six hours over an distance of 536 km. Cattle were received in shipments over three separate days and housed overnight in feedlot receiving pens with hay and water available. Processing occurred the following day and cattle were allocated to their respective pens. In some cases, initial processing did not occur until two days post arrival due to time limitations relating to processing. A summary of the processing durations by animal and day are given in Table 3.3. Processing time per animal was 111.92 s in year one and 97.94 s per animal in year two. An average of 310 and 264 head were processed each day in year one and two, respectively. There were an average of 260 head per pen (6 pens in 2007 and 5 pens in 2008) with a minimum of 99 and a maximum of 310 head per pen (Table 3.4). In year one, pens were dispersed among mixed source cattle upon arrival. In year two the

commercial feedlot was not at capacity and there was no fence line contact with outside cattle until later in the finishing period.

Year 1 (2007):

Processing entailed radio frequency identification tag, visual identification tag, lot tag, 30 ml of blood, ultrasound measurements of loin eye muscle area, backfat, and percent intramuscular fat, temperament measurements of chute score and exit velocity (BIF, 2002), tissue sample for persistently infected bovine viral diarrhea virus (**BVD-PI**) tests, and a hair sample. Rumen temperature boluses were administered to two pens of steers. All animals received an oral and pour on parasiticide, Synanthic and Promectin, respectively along with a growth promotant, Revalor-IS. No antimicrobials were administered during the first year to ensure a sufficient disease (BRD) challenge within the population.

Year 2 (2008):

The same phenotypes were collected in year two as in year one with the addition of rumen boluses to all animals (n=1,319). Parasticides included an oral, pour on and injectable, Safeguard, Promectin, and Normectin, respectively. Steers were implanted with a 200 d delayed releasing growth promotant Revalor XS. Steers in year two received antimicrobials upon arrival, Pyramid 2 + Type II BVD and Presponse SQ, against BVD, *Pasteurella multocida* and *Mannheimia haemolytica*, common viral and bacterial agents associated with BRD.

Table 3.3. Summary of processing time

	MEAN	SD	MIN[1]	MAX[1]
Processing time[2]				
2007	111.92	51.92	45.30	577.54
2008	97.94	35.23	51.47	451.59
Total	105.52	45.58	45.30	577.54
No. Processed per d.				
2007	310	59.26	220	382
2008	264	64.95	200	351
Total	287	63.55	200	382

[1]Min = Minimum value; Max = Maximum Value

[2] Processing time in seconds

Table 3.4. Number of steers per pen by year

	Pen					
Year	1	2	3	4	5	6
2007	310	305	217	310	310	99
2008	290	272	205	276	276	-

3.3 Health Management

Health of the steers was monitored daily by commercial feedlot's personnel. Pens were ridden once per day in the morning. Steers were removed for treatment by the cowboys based on the commercial feedlots protocols for identification of sick and lame animals. Once pulled, steers were moved across the alley onto the Colorado State

University South Eastern Colorado Research Center (**SECRC**) for evaluation. At that time a list of clinical symptoms were evaluated; lethargy, depression (droopy ears), nasal and optical discharge, cough, and rectal temperatures over 103.5^{o} F, to determine BRD infection. A steer presenting with multiple clinical symptoms and/or a rectal temperature greater than 103.5^{o} F were assumed to be affected by BRD. Treatment protocols were administered accordingly for animals showing clinical signs of BRD, lameness, pinkeye, and bloat as set by the commercial feedlot and SECRC standard operating procedures. Steers were housed in ten head pens at the SECRC facility. Infected steers were monitored daily evaluating the list of clinical symptoms described earlier, weight, and rectal temperature. Steers with a rectal temperature below 103.5 ^{o} F between day 5 and 7 post treatment were considered healthy and returned to their respective pens in the commercial feedlot. Steers continually temping above 103.5^{o} F by day 7 received a second BRD treatment. Steers were continually monitored for 5 d and reevaluated, and treated as necessary in 5 d increments if rectal temperature was greater than 103.5 ^{o}F.

3.4 Lung Lesion Scoring at Harvest

Cattle were harvested at JBS Swift and Company plants in Dumas, TX and Greeley, CO in years one and two, respectively. Relevant carcass and lung lesions scores were collected. A modified system described by Bryant et al., (1999) was implemented for the collection of lung lesion scores at chain speed. The system scores on a scale of 0 to 3 where a score of 0 indicates normal lung which may include depressed healed areas; a score of 1 has lung damage in less than 5% of the total lung volume, minimal adhesions or pleuritis (fibrin tags); a score of 2 indicates numerous adhesions affecting more than one anterior ventral lobe, and a score of 3 had large amount of missing lung tissue caused

by pleuritis and/or adhesions greater than 15% of the total lung volume, or active lymph nodes indicating a current infection. Lungs were scored concurrently by two trained evaluators and averaged to obtain a single mean lung score.

3.5 Literature Cited

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4 Characterization of bovine respiratory disease prevalence in feedlot steers and associated effects on carcass performance[12]

A paper to be submitted to the Journal of Animal Science

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4.1 Abstract

ABSTRACT: Bovine respiratory disease (**BRD**) is one of the most prevalent and economically limiting diseases facing the United States beef industry today. Therefore, the objectives of this study were to (1) characterize the occurrence and prevalence of BRD in feedlot steers utilizing three disease classifications; treatment records (**Trt**), lung lesions present at harvest, and total BRD (treatment or lung lesion or both), and (2) to examine the effects of Trt, number of treatments (**NumTrt**), mean lung score, lesions present, and total BRD on economically relevant carcass traits. Data included health and carcass records on 2,870 crossbred steers managed in a commercial feedlot in Southeast Colorado over a two year period. Disease prevalence varied from 45% (n = 698) to 7% (n = 94) in years one and two, respectively. Averaged across the two year period, 27.6% of the steers exhibited clinical signs and were treated for BRD. Lung lesions were collected on 1,226 and 1,261 steers in year one and year two, respectively. Lesions were present in 71% in of steers year one and 47% in year two, and were present in 59% (n=1,461) of the steers over the two year period. Incidence for total BRD, was 76%, 59%, and 64% for years one, two, and overall, respectively. A subset of data (n = 1,260) of animals with birth information was used to evaluated the effect of age ($P > 0.05$) and BRD classification on carcass traits. Similarly, two models were fitted to the complete data to test the effect of receiving BW as an approximation for age. Evaluation of the models indicated receiving weight to be a significant fixed effect in prediction carcass merit ($P < 0.001$). Incidence of BRD in the feedlot had a negative impact on both marbling score (**MS**) and subcutaneous backfat thickness (**Fat**) ($P < 0.001$), while accounting for minimal variation ($P > 0.05$) in HCW and LM area after adjusting for fixed contemporary

group effects and receiving BW. However, animals that were chronically infected (at least 3 treatments) had reduced HCW (-16.5 ± 4.5 kg) and LM area (4.9 ± 1.25) ($P < 0.05$) compared to steers that were never treated. Lesions present at harvest did not have a significant effect ($P > 0.05$) on any evaluated traits. Animals that were categorized as suffering from BRD by the definition of total BRD had reduced MS (6.1 ± 2.8) and Fat (0.39 ± 0.18) ($P < 0.05$) when compared to healthy steers.

Key words: bovine respiratory disease, lung lesions, beef cattle

4.2 Introduction

Disease prevalence has become a growing concern for beef cattle producers and consumers alike. The influx of branded and natural beef products illustrates the conciseness of consumers toward healthy and healthful beef products. Bovine respiratory disease (**BRD**) is the most prevalent and economically crippling disease facing the beef cattle industry (USDA NAHMS, 2000). Economic losses from postweaning incidences of BRD have been estimated to range from $15.57 to $92.26 per head for treated animals (McNeill et al., 1996; Faber et al., 1999; Schneider et al., 2009).

Bovine respiratory disease results from complex interactions between physical, environmental, and epidemiological factors, including both viral and bacterial pathogens (Callan and Garry, 2002). The multifaceted nature of BRD increases the complexity of prevention management because disease incidence and outbreaks cannot be attributed to a single causative agent. Evaluation of disease effects are often biased because diagnosis of infection may be imprecise. It is often difficult to identify when the infection occurred,

causation of the infection, immune status of individuals, and whether or not clinically normal animals were adequately exposed (Snowder 2006a). Lung lesions present at slaughter have been reported to identify underlying prevalence in feedlot cattle (Wittum et al., 1996; Bryant et al., 1999; Thompson et al., 2006) better than feedlot treatment records alone.

The first objective of this study was to characterize the occurrence and prevalence of BRD in feedlot steers utilizing three disease classifications; treatment records (**Trt**), lung lesions present, and total BRD (treatment or lung lesions or both). The second objective was to examine the effects of the disease classifications on economically relevant carcass traits.

4.3 Materials and Methods

All experimental procedures were reviewed and accepted by the Colorado State University Animal Care and Use Committee (07-230A-01).

Crossbred steer calves were obtained from a single ranch source in western Nebraska originating from one of three units in November of 2007 and 2008 (n = 1,551 and 1,319; respectively) and shipped to a commercial feedlot in Southeastern Colorado. Cattle were in transit approximately seven hours over a distance of 536 kilometers. Each year, steers were received over three days and housed overnight in feedlot receiving pens with unlimited access to grass hay and water. Processing occurred the following day and cattle were allocated to their respective pens. In some cases, initial processing did not occur until two days post arrival due to time limitations related to processing length.

Once processed, steers were housed in pens averaging 260 head per pen (6 pens in 2007 and 5 pens in 2008) with a minimum of 99 and a maximum of 310 head per pen.

Year 1 (2007)

Processing entailed application of radio frequency identification tag, visual identification tag, and lot tag; collection of 30 ml of blood, a tissue sample for identification of animals persistently infected with bovine viral diarrhea, and a hair sample; ultrasound measurements of loin eye muscle area, backfat, and percent intramuscular fat; temperament measurements of chute score and exit velocity;. Rumen temperature-sensing boluses were administered to two pens of steers. All animals received oral and pour-on parasiticides, Synanthic and Promectin, respectively along with a growth promotant, Revalor-IS. No vaccinations were administered during the first year to ensure a sufficient disease (BRD) challenge within the population.

Year 2 (2008)

The same phenotypes were collected in year two as in year one with the addition of application of rumen temperature sensing boluses to all animals (n=1,319), of parasticides including an oral, pour on and injectable, Safeguard, Promectin, and Normectin, respectively. Steers were implanted with a 200d delayed releasing growth promotant Revalor XS. Steers in year two received vaccination upon arrival to the feedlot, Pyramid 2 + Type II BVD and Presponse SQ, were against BVD and *Pasteurella multocida* and *Manheimia haemolytica*, common viral and bacterial agents associated with BRD.

4.3.1 Feedlot Health

Health of the steers was monitored daily by the commercial feedlot's personnel to identify individuals with clinical signs of disease and followed the commercial feedlots protocols for identification of sick and lame animals. Steers determined to be diseased or lame by feedlot personnel were removed from their respective pens and transferred to the adjacent Colorado State University South Eastern Colorado Research Center (**SECRC**) for further evaluation by researchers. Individuals were then evaluated for clinical symptoms of BRD including; lethargy, depression, nasal and optical discharge, and cough. In addition rectal temperatures were collected to aid in determination of BRD infection. A steer exhibiting multiple clinical symptoms, or a rectal temperature greater than 103.5^{o}F, or any combination thereof, were determined to be affected by BRD.

Treatment protocols were administered according to standard operating procedures of the SECRC and the commercial feedlot appropriate for animals suffering from BRD, lameness, pinkeye, or bloat. After treatment, steers were housed in ten head pens on the SECRC facility until time of recovery, from determined BRD ailment. Steers with a rectal temperature below 103.5 ^{o} F and displaying no combination of clinical symptoms between day 5 and 7 post treatment were considered recovered and returned to their respective pens in the commercial feedlot. Steers with rectal temperatures above 103.5^{o}F or exhibiting multiple clinical symptoms by day 7 of the initial treatment received a second BRD treatment. Steers were continually monitored for 5 d and reevaluated, and treated as necessary in 5 d increments when presenting with a rectal temperature greater than 103.5 ^{o}F. Animals identified by feedlot personnel that had been

returned from a previous hospital visit continued on a protocol from the time of last treatment record.

4.3.2 Carcass and lung lesion data

Cattle were humanely harvested at JBS Swift and Company plants in Dumas, TX and Greeley, CO in years one and two, respectively. Relevant carcass and lung lesions scores were collected by trained personnel. Carcass traits included HCW, LM area, marbling score (**MS**), and subcutaneous backfat thickness (**Fat**). Counts of valid carcass observations are given in Table 4.1.

A modified system described by Bryant et al. (1999) was implemented for the collection of lung lesion scores at commercial plant chain speed. The system scores lungs on a scale of 0 to 3 where a score of 0 is indicative of normal lung which may include depressed healed areas; a score of 1 has lung damage in less than 5% of the total lung volume, minimal adhesions or pleuritis (fibrin tags); a score of 2 indicates numerous adhesions affecting more than one anterior ventral lobe, with a score of 3 having large amounts of missing lung tissue caused by pleuritis and/or adhesions greater than 15% of the total lung volume, or active lymph nodes indicating a current infection. Lungs were scored concurrently by two trained evaluators and averaged to obtain a single lung score.

4.3.3 Statistical Analysis

Incidence of BRD, measured as Trt, was binary with a classification of 0 indicating a clinically healthy animal and a classification of 1 indicating an individual that was treated for BRD at least once during the feeding period. The total number of BRD treatments (**NoBRD**) an animal received over the duration of the feeding period

was defined as: 0 for animals never treated, 1 and 2 for animals treated once and twice, respectively, and 3 for chronically infected animals treated thrice or more. Lung lesion data were evaluated using two different approaches. A mean lung lesion score from two trained evaluators was calculated to estimate lung lesion severity. Alternatively, lung lesions scores were collapsed into a binary outcome, with 0 indicating no measurable lesions present or minimal lung damage (mean lung score < 1.5) and 1 having lesions present (mean lung score ≥ 1.5). Total BRD was defined as binary and categorized by a combination of BRD and lung lesions. A steer with an observation of 1 had either a 1 for a Trt observation or a 1 for lung lesions present, or both, indicated an animal suffering from BRD. A classification of 0 indicated a clinically healthy animal that had no observed Trt (0) or lesion present (0).

Cluster analysis was investigated as an alternative approach for classifying diseased versus healthy animals. The goal of the cluster analysis was to identifying homogeneous subgroups based on treatment, mean lung score, and feedlot performance. Severity of lung lesions present at harvest have been shown to have a high association with reduction in ADG over the feeding period, particularly in the first few weeks after arrival to the feedlot when the highest BRD incidence is observed (Wittum et al., 1996; Bryant et al., 1999; Thompson et al., 2006). Data for the analysis included Trt, mean lung lesion score, and early average daily gain (**EADG**) and overall average daily gain (**OADG**). A subset of data for animals with valid records for all evaluated traits was used (n = 2,434) to calculate Euclidean distances. The analysis was performed using k-means procedure from the cluster package in R (R Development Core Team, 2008) which implements Euclidean distance to form cluster groups. Kendal rank correlations were

then used to estimate re-ranking of animals between cluster groups and both Trt, and lesions present classifications.

A subset of data (n = 1,260) of steers with birth date information were evaluated to determine the fixed effect of age and BRD classification on evaluated carcass measurements. Numbers of carcass records for the subset data are presented in (Table 4.2). The MIXED procedure (SAS Inst. Inc., Cary, NC) was used to estimate effects of Trt, NoBRD, lung score, lesions present, and total BRD on carcass performance. The regression equation used for all outcomes can be described as:

$$y_{ijk} = BRD_j + CG_k + Age_i + An_{ijk} + e_{ijk}$$

Where: y_{ijk} is random continuous carcass trait measured on the i^{th} steer, with BRD treatment classification j, with j = Trt, NoBRD, mean lung score, lesions present, or total BRD ; CG_k is the fixed effect of the k^{th} contemporary group defined as ranch unit, feedlot pen, year, and harvest date combinations; Age_i is the linear covariate of age at harvest, and both An_{ijk} and e_{ijk} are random terms relating to animal and residual error, respectively. It random terms of animal and residual error were assumed to be distributed as An ~ N(0, σ_{An}^2) and e_{ijk} ~ N(0,σ_e^2).

The complete data were evaluated using two models. The first model (**M2**) contained receiving BW as a linear covariate for an approximation for age. Due to the high correlation of BW with traits such as HCW and LM area, a reduced model (**M2**) was also fit with CG and BRD classification as the only fixed effects. Models were compared using Akaike's information criterion (AIC) to evaluate best fit.

4.4 Results and Discussion

4.4.1 Characteristics of BRD Classifications

Treatment records

From the subset of data with birth and weaning information the average age at receiving ranged from 160 to 270 d of age. The mean receiving weight of the entire population was 221 (SD = 24.3), 223.9 (SD = 23.5), and 229.1 (SD = 25.1) kg for all animals, year one animals, and year two animals respectively.

Number of animals treated by day over the duration of the feeding period for the two years is illustrated in Figure 4.1. The peak incidence occurred on d 13 and 10 for years one and two, respectively. The vast majority of BRD was experienced in the first 35d of the feed phase at 75% and 60% of treatments of year one and year two, respectively. These results are in agreement with typical treatment rates in the first weeks after arrival into the feedlot (Wilson et al., 1985; Bateman et al., 1990; Thompson et al., 2006, Babcock et al., 2008). During year one a total of 698 steers (45.0%) were treated for BRD, 94 steers (7.0%) in year two, and 792 total (27.6) over the combined years. Of the treated animals in year one 177 (28.5%) and 62 (8.9%) were treated two and three times, while in year two 4 (4.3%) and 5 (5.3%) were treated twice or thrice. Incidence of BRD was much higher in year one than two and illustrates the variability of disease incidence and managerial practices over sequential years also shown by Snowder et al. (2006b). However, differences in disease prevalence could not be adjusted for effect of vaccination upon arrival to the feed lot in subsequent years. Incidence rates for year two were similar to the 8.7% incidence rate reported by Schneider et al. (2009), which implemented a standard vaccination protocol across several commercial feed lots.

Lung Lesions

Lung lesion scores were collected at harvest on 1,226 and 1,262 steers for years one and two, respectively and a total of 2,487 steers over the combined years. Lesions were present in 70.7% of evaluated lungs in year one and 47.1 % in year two for a combined rate of 58.8% (n=1,461) of all animals (Table 4.3). The total percentage of cattle with lesions present was similar to the 61.9% reported by Schneider et al. (2009) and higher than the 42.8% reported by Thompson et al. (2006) and lower than the 72% reported by Wittum et al., (1996). Distributions of animals treated and presence of lung lesions at harvest are given in Table 4.2. Of the 617 total steers treated, 68.9 % had lung lesions present at harvest. The data herein also shows that there are animals that suffer from subclinical symptoms of BRD as 70.2% of the steers that were not treated had lesions present at harvest in year one and 46.8% in year two, with 55.4% over the two years combined. These apparently high percentages of misclassified animals identified through the comparison of morbidity records and lung lesions illustrate the inherent complications that arise while evaluating disease field data (Table 4.3). Reasons for misclassification may be due to steers were wrongly identified as being affected with BRD during the feeding period, steers that were treated recovered before sufficient damage occurred to the lungs, or steers with presenting with lesions but were never treated may have suffered from BRD earlier in life.

Total BRD

Due to feedlot mortality and premature culling of chronically infected animals preharvest, lung lesions fail to encompass the entire population. Therefore, Trt and lung

lesion presence were combined to evaluate total BRD prevalence. An individual steer was classified as diseased if it had an observable treatment record during the feeding phase or had observable lung lesions at harvest, or both. Individuals not treated without any subsequent lung lesions at harvest were categorized as healthy. In year one 76.1% (1,181) of steers were classified as diseased, and 49.1% (657) in year two, and 63.7% (1,828) over both years.

Comparisons of BRD prevalence between the classifications of BRD (treatment records, lung lesions, and total) are given in Table 4.4. Prevalence of BRD increased with the inclusion of lung lesions when compared to treatment records alone as previously reported by Wittum et al., 1996, Bryant et al., 1999, Thompson et al., 2006, Schneider et al., 2009).

Cluster Classification

Due to potential misclassification of diseased animals in the classification methods described above, cluster analysis was performed to evaluate an alternative method for identifying infected animals. Cluster 1 (**C1**) grouped animals based on treatment records (yes/no), mean lung score (0-3) and overall feeding period ADG. The prevalence of disease in C1 was estimated at 39.2%, reclassifying 725 (29.8%) steers between Trt and C1 and 477 (19.6%) steers between lung score present and C1. Kendall rank correlations between Trt and C1 and lung score and C1 were 0.35 ($P < 0.001$) and 0.67 ($P < 0.001$), respectively. The first two principal components explained 72.94% of the point variation (Figure 4.2) associated with the C1 classification of diseased and healthy animals. Cluster 2 (**C2**) was created by replacing OADG with EADG, which was

72 d and 97 d post arrival for year one and two, respectively. Disease prevalence of C2 was estimated to be 58.6%, reclassifying 1,202 (49.4%) steers from Trt with a rank correlation of 0.12. No steers were reclassified between the binary classification of lesions present and the C2 classification (correlation = 1; P-value < 0.001). The first two PC from the C2 analysis explained 74.8% (Figure 4.3) of the point variation between diseased and healthy steers as classified by C2. The amount of point variation explained in C2 was greater than that of C1 and was determined to estimate diseased animals better than C1. This finding is in agreement with previous research that lung scores have a high association with EADG (Wittum et al., 1996; Bryant et al., 1999; Thompson et al., 2006). Because C2 had a perfect correlation with lesions present, it was assumed that they were the same trait, and was not evaluated in further analysis.

4.4.2 Carcass Traits

Subset data

A summary of carcass traits for data subset for age at harvest is presented in Table 4.2. Significance of fixed sources of variation are presented in Table 4.5. Harvest age was not found to be significant in predicting carcass traits with the exception of MS in all models. Steers that were treated for BRD during the feeding period had lighter HCW (8.01 ± 2.48 kg; P-value < 0.001) as well as less Fat (0.69 ± 0.27 mm; P-value < 0.029) (Table 4.6). There was also a decrease in HCW as the frequency of BRD treatments increasing when compared to steers that never received a treatment. Mean lung score severity had higher least square mean estimates for all levels for all traits compared to animals with a "0". Similarly steers with lesions present had 1.5 ± 0.51cm^2 less LM area

(P-value < 0.038) than steers with no lesions present. Biologically the results for mean lung score and lesion present are not understood, as performance would be expected to decrease with increasing severity of lung lesions, and may be an artifact of the subset data.

Complete data

A summary of carcass performance is presented in Table 4.1. Significance of fixed sources of variation and for M1 and M2 are given in Table 4.7. All BRD classifications were significant for the same carcass traits between models except for the effect of Trt on HCW and mean lung score on Fat. The full model (M1), which included receiving BW, was better at predicting carcass traits based on AIC (Table 4.7). Receiving BW was significant for all evaluated carcass traits, excluding MS. Least square means for carcass traits where BRD classification was found to be significant for M1 and M2 are similar in magnitude and are presented in Table 4.8 and Table 4.9, respectively

Steers receiving treatment for BRD during the feeding period had lower MS (11.31 ± 3.31; P-value < <0.001) and Fat (0.62 ± 0.22 mm; P-value < 0.003) when compared to those that had never been treated. The current estimates are similar to those reported by Schneider et al., (MS = 13 ± 4.0 and FAT = 0.76 ± 0.25 mm). Steers that were considered chronically infected (treated 3 or more times) had lighter HCW (-16.5 ± 4.49 kg;P < 0.001) and smaller LM area (5.56 ± 1.28; P-value < 0.001) .The current estimate for HCW is more than the -9.2 estimate of Roeber et al. (2001) for animals treated twice or more. Similarly steers treated once or twice had less MS and FAT than those that were never treated during the feeding period. These results agree with both

Roeber et al. (2001) and Gardner et al. (1999) who illustrated a decrease in both MS and Fat with increasing number of treatments.

Presence of lesions at harvest did not have a significant effect (P-value > 0.05) on any of the evaluated carcass traits. This result is in agreement with reports of Schneider et al. (2009) on the effect of lesion presence on carcass traits; however, disagrees with the report of Gardner et al. (1999) who reported a decrease in MS with the presence of lung lesions. Mean lung score were associated with an increase in performance for both HCW and MS (Table 4.8). These results may be an artifact of the current data set and suggest further investigation with other populations. Bryant et al. (1999) warns that only lung lesions resulting from cranial ventral bronchopneumonia were significantly associated with decreasing performance. Though several lesion types may exist, they have no association with decreased performance. The lack of evidence in the current study for presence of lesions and severity may indicate that evaluators misclassified lesion types and scored animals not suffering from cranial ventral bronchopneumonia lesions alone.

The combination of Trt and lesions present into total BRD was significant in predicting both MS and Fat ($P \leq 0.05$). Animals categorized as diseased by this definition had reduced MS (6.19 ± 2.82; P-value < 0.03) and Fat (0.39 ± 0.18; P-value < 0.03). Total BRD did not have a significant effect on HCW or LM area (P> 0.05) which contradicts the -3.0 ± 1.73 and -0.65 ± 0.39 estimate for diseased animals reported by Schneider et al. (2009) for HCW and LM area, respectively.

4.5 Implications

Results from this study indicate that BRD morbidity experienced during the finishing phase has a negative impact on carcass quality. Decreased performance relating to BRD treatments illustrates the importance for prevention and treatment protocols to be implemented in commercial feedlots. It can be extrapolated that these decreases in carcass quality due to morbidity will have negative impacts on profitability especially when cattle are marketed on a grid or as a natural or branded product. With the direct and indirect costs associated with disease incidence, the need for a tool in which producers may select for animals that are less susceptible to BRD becomes apparent.

Contrary to previous results, lesions present at harvest had no apparent effect on carcass quality while the severities of lesions was associated with an increase in performance. The discrepancy may be due to misclassification of lesions observed that are not truly associated with a decrease in performance. The potential for misclassification of lesion types at chain speed brings into question the reliability of lesions at harvest as an accurate measure to truly predict disease prevalence.

4.6 Literature Cited

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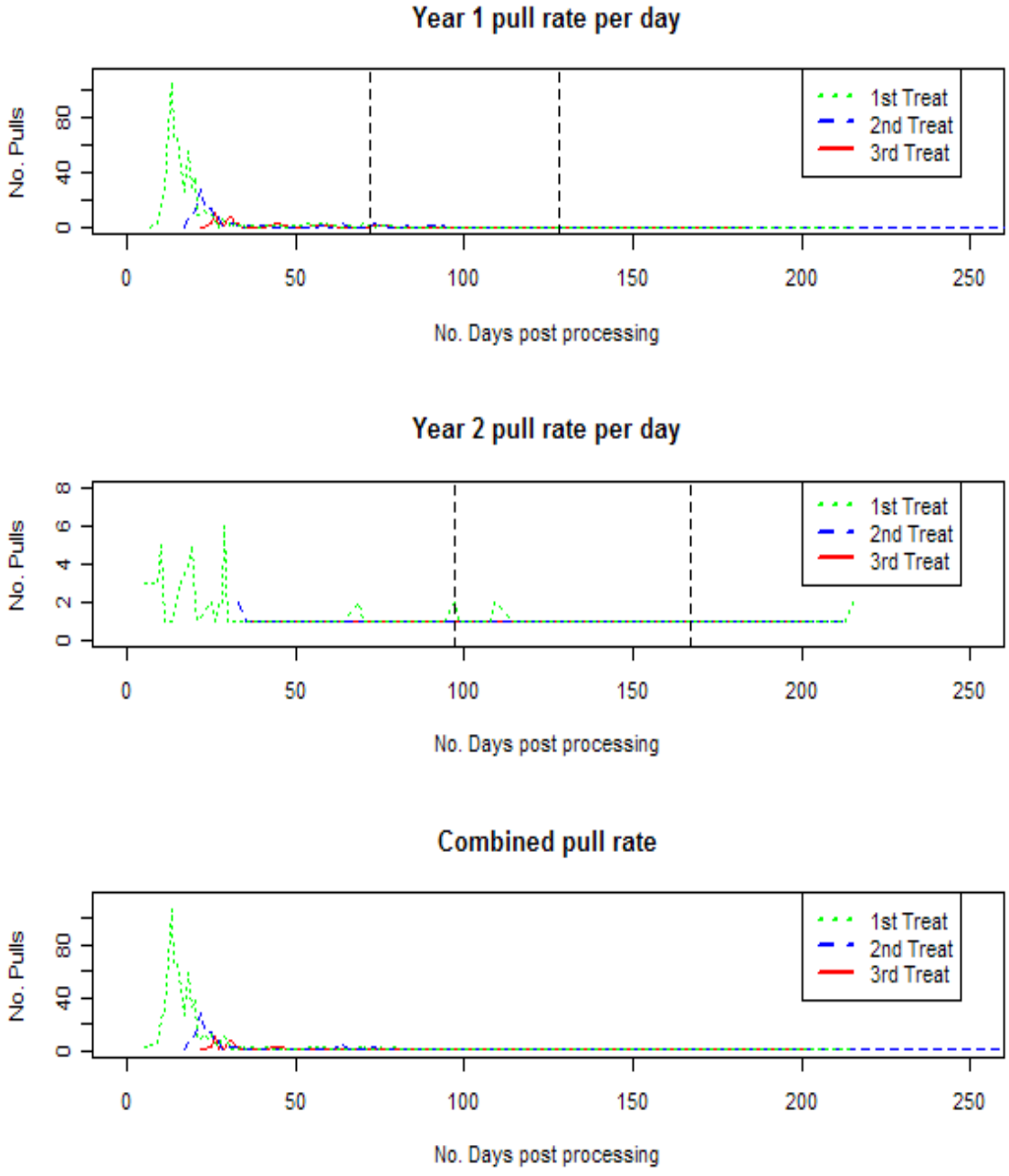
Figure 4.1 Treatment rates per day for year one (2007) year two (2008) and combined[1]



[1]Vertical lines in year 1 and year 2 graphs indicate subsequent processing days

Table 4.1. Summary statistics for evaluated carcass traits[1] of treated and non-treated feedlot steers

	n	Mean	SD	Minimum	Maximum
HCW	2,525	354.4	32.9	154.7	474.0
LM area	2,518	82.2	8.3	50.7	115.8
MS	2,524	403.2	65.2	250	750
FAT	2,516	13.0	4.2	0.8	36.6

[1] HCW = hot carcass weight (kg), LM area = longissimus muscle area (cm^2), MS = marbling score, Fat = subcutaneous backfat thickness (mm)

Table 4.2. Summary statistics for subset carcass data[1]

	n	Mean	SD	Minimum	Maximum
Harvest age	1,260	450.5	18.3	390	502
HCW	1,259	352.4	32.8	164.7	474
LM area	1,255	81.7	8.4	56.7	115.8
MS	1,258	412.7	65.7	250	750
Fat	1,253	13.1	4.1	0.8	36

[1]Harvest age = d, HCW = hot carcass weight (kg), LM area = longissimus muscle area (cm^2), MS = marbling score, Fat = subcutaneous backfat thickness (mm)

Table 4.3. Distribution as a percentage of total number of steers treated for bovine respiratory disease (BRD) and lesions present at harvest

		No. (%) of Steers			
Year	No. of steers	Treated for BRD	Lesions present at harvest	Treated and Lesions	Not treated and lesions
2007	1,226	538 (43.9)	867 (70.7)	384 (71.4)	483 (70.2)
2008	1,261	79 (6.3)	594 (47.1)	41 (51.9)	553 (46.8)
Combined	2,487	617 (24.8)	1,461 (58.8)	425 (68.9)	1,036 (55.4)

Table 4.4. Bovine respiratory disease (BRD) prevalence and number of observations based on disease classification[1]

	Year 1 (2007)		Year 2 (2008)		Combined	
	Total No.	No. (%) Disease classification	Total No.	No. (%) Disease classification	Total No.	No. (%) Disease classification
Trt	1,551		1,319		2,870	
0		853 (55.0)		1,225 (93.0)		2,078 (72.4)
1		698 (45.0)		94 (7.1)		792 (27.6)
NumTrt	1,551		1,319		2,870	
0		853 (55.0)		1,225 (93.0)		2,078 (72.4)
1		520 (33.5)		85 (6.3)		605 (21.1)
2		117 (7.5)		4(0.3)		121 (4.2)
3		61 (3.9)		5 (0.4)		66 (2.3)
Mean lung score	1,226		1,261		2,487	
0		119 (9.7)		41 (3.3)		160 (6.4)
$0 < i \leq 1$		75 (6.2)		306 (24.3)		381 (15.3)
$1 < i \leq 2$		388 (31.6)		708 (56.1)		1,096 (44.1)
$2 < i \leq 3$		644 (52.5)		206 (16.3)		850 (34.2)
Lesions Present	1,226		1,261		2,487	
0		359 (29.3)		667 (53.0)		1,026 (41.3)
1		867 (70.7)		594 (47.0)		1,461 (58.7)
Total BRD	1,551		1,319		2,870	
0		370 (23.9)		662 (50.1)		1,042 (36.3)
1		1,181 (76.1)		657 (49.1)		1,828 (63.7)

[1]Trt = BRD incidence ,NumTrt = number of BRD treatments, Mean lung score = average of two lung lesion scores, Lesions present = binary (yes/no), Total BRD = binary (yes/no) for Trt or Lesions present, or both.

Figure 4.2. Plot of first two principal components against k-means cluster of bovine respiratory disease treatment (BRD), lung lesion score, and overall ADG.

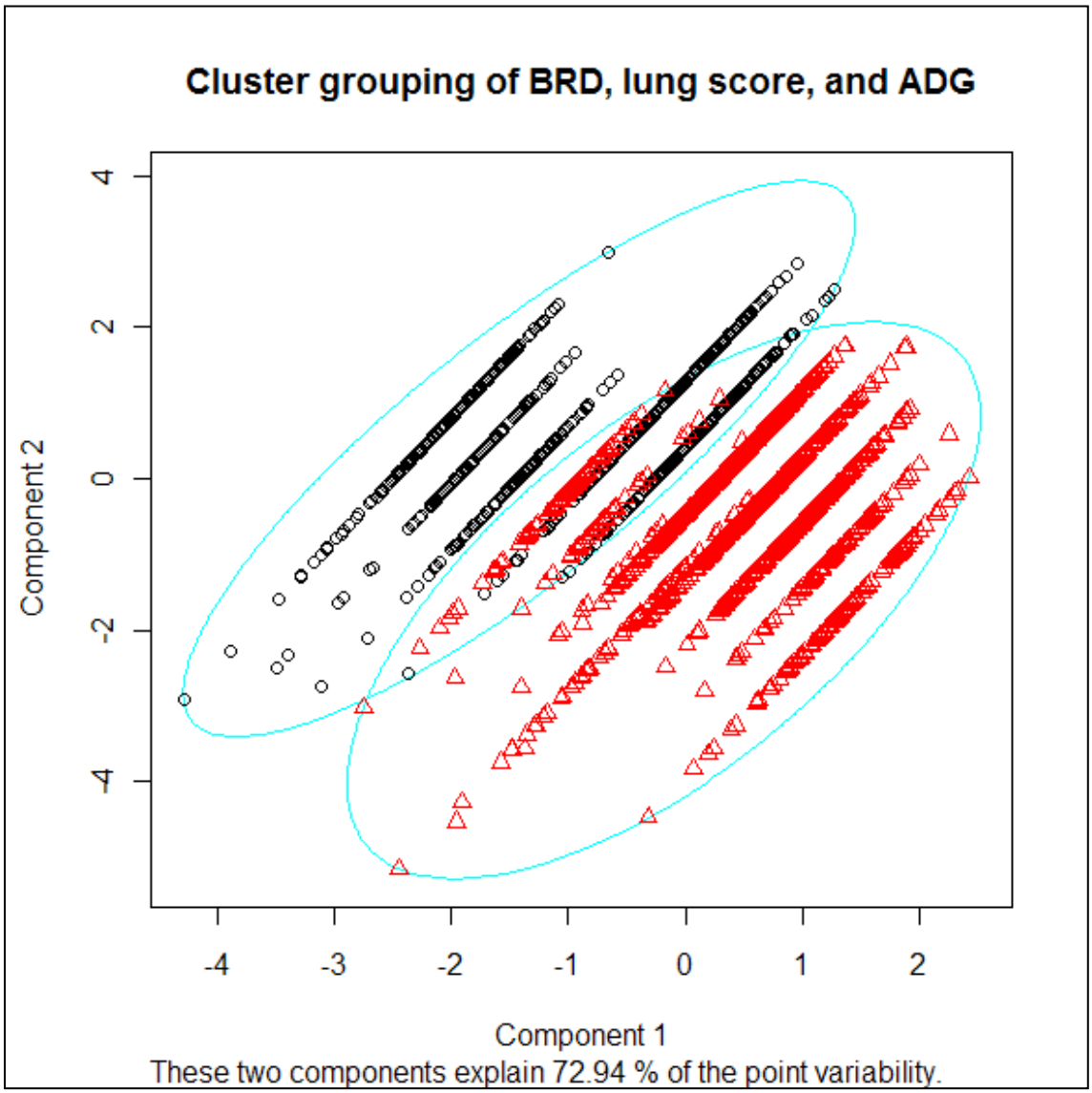


Figure 4.3. Plot of first two principal components against k-means cluster of bovine respiratory disease treatment (BRD), lung lesion score, and early average daily gain.

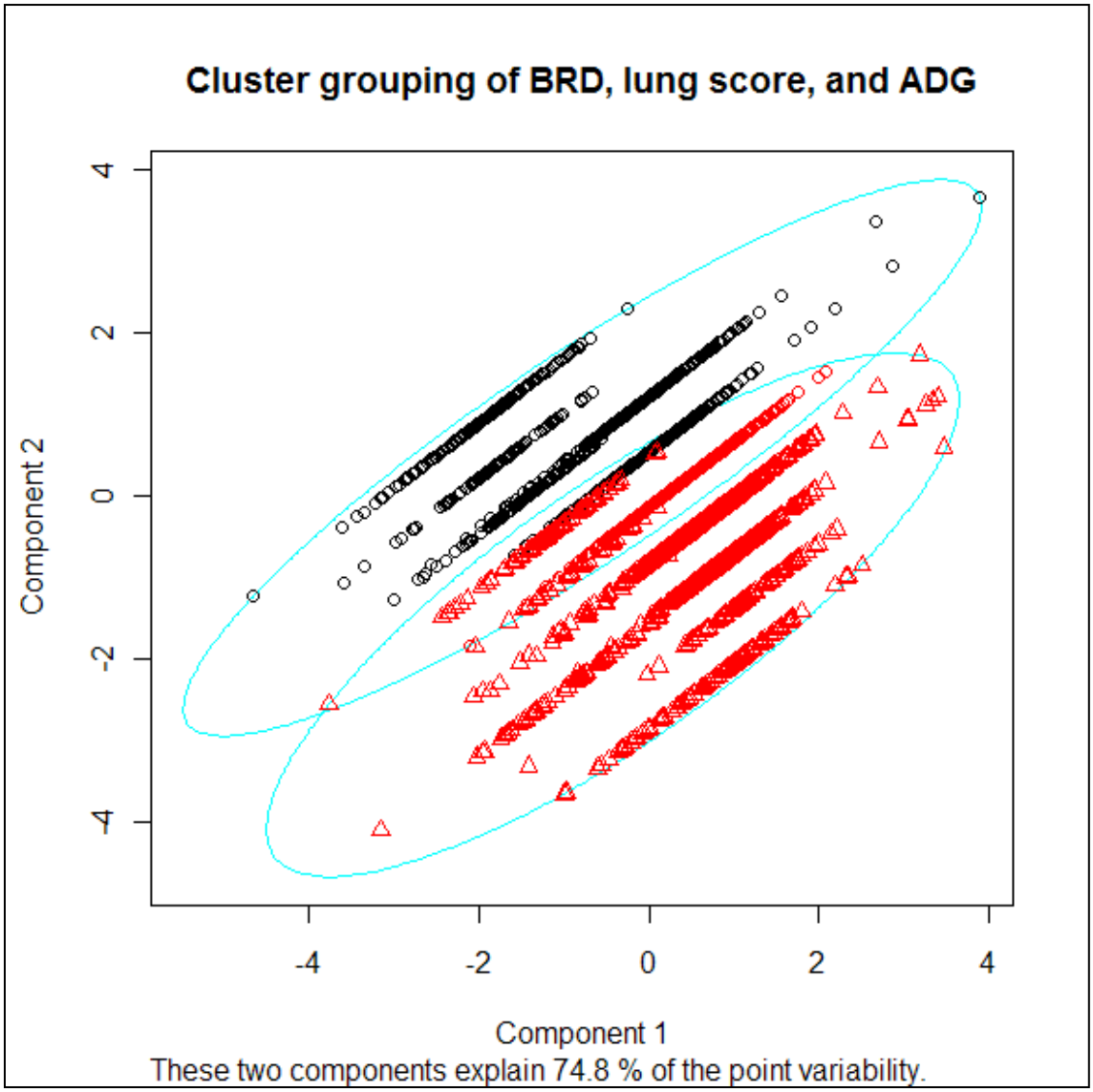


Table 4.5. Significance of fixed sources of variation for carcass traits[1] in feedlot cattle by bovine respiratory disease (BRD) classification[2] for data subset for age at harvest

			P-value[3]		
		n	CG	Age	BRD
Trt					
	HCW	1,259	<0.001	0.124	0.001
	LM area	1,255	<0.001	0.223	0.087
	MS	1,258	<0.001	<0.001	0.068
	Fat	1,253	<0.001	0.971	0.029
NumTrt					
	HCW	1,259	<0.001	0.117	0.009
	LM area	1,255	<0.001	0.244	0.166
	MS	1,258	<0.001	<0.001	0.153
	Fat	1,253	<0.001	0.959	0.086
Mean lung score					
	HCW	1,259	<0.001	0.032	0.005
	LM area	1,255	<0.001	0.277	0.142
	MS	1,258	<0.001	<0.001	0.008
	Fat	1,253	<0.001	0.682	0.011
Lesions present					
	HCW	1,259	<0.001	0.031	0.765
	LM area	1,255	<0.001	0.222	0.038
	MS	1,258	<0.001	<0.001	0.553
	Fat	1,253	<0.001	0.7	0.636
Total BRD					
	HCW	1,259	<0.001	0.063	0.795
	LM area	1,255	<0.001	0.114	0.338
	MS	1,258	<0.001	<0.001	<0.001
	Fat	1,253	<0.001	0.82	0.147

[1]Trt = BRD incidence ,NumTrt = number of BRD treatments, Mean lung score = average of two lung lesion scores, Lesions present = binary (yes/no), Total BRD = binary (yes/no) for Trt or Lesions present, or both.
[2]HCW = hot carcass weight (kg), LM area = longissimus muscle area (cm^2), MS = marbling score, Fat = subcutaneous backfat thickness (mm)
[3]effect is signficicant at $P < 0.05$

Table 4.6. Least squares means (± SE) for bovine respiratory disease (BRD) classification[1] effect on carcass traits[2] subset for harvest age

	Classification level						
	0	0.5	1	1.5	2	2.5	3
Trt							
HCW	357.18 (1.23)	-	-8.01 (2.48)*	-	-	-	-
Fat	13.54 (0.16)	-	-0.69 (0.27)*	-	-	-	-
NumTrt							
HCW	357.19 (1.23)	-	-7.06 (2.65)*	-	-11.39 (5.52)*	-	-14.83 (8.92)
Mean lung score							
HCW	344.16 (3.83)	20.04 (5.99)*	18.34 (4.82)*	9.49 (4.40)*	11.92 (4.28)*	11.74 (4.40)*	11.11 (4.36)*
MS	394.43 (7.70)	16.36 (12.06)	37.15 (9.69)*	18.65 (8.84)*	18.96 (8.62)*	13.17 (8.86)*	19.46 (8.62)*
Fat	11.73 (0.49)	2.45 (0.77)*	1.95 (0.62)*	1.51 (0.56)*	1.63 (0.55)*	1.23 (0.56)*	0.85 (0.56)
Lesions present							
LM area	81.83 (0.41)	-	1.05 (0.51)*	-	-	-	-
Total BRD							
MS	415.83 (3.52)	-	-4.58 (4.11)	-	-	-	-

[1]Trt = BRD incidence ,NumTrt = number of BRD treatments, Mean lung score = average of two lung lesion scores, Lesions present = binary (yes/no), Total BRD = binary (yes/no) for Trt or Lesions present, or both.
[2]HCW = hot carcass weight (kg), LM area = longissimus muscle area (cm^2), MS = marbling score, Fat = subcutaneous backfat thickness (mm)
* LSMEAN is different from animals that have never been treated (0) $P<0.05$

Table 4.7. Significance of fixed sources of variation for carcass traits[1] in feedlot cattle by bovine respiratory disease (BRD) classification[2]

	Full model (M1)					Reduced model (M2)		
			P-value				P-value	
	n	AIC	CG	BW	BRD	AIC	CG	BRD
Trt								
HCW	2,525	23,979	<0.001	<0.001	0.104	24,637	<0.001	0.011
LM area	2,518	17,572	<0.001	<0.001	0.888	17,681	<0.001	0.785
MS	2,524	27,879	<0.001	0.964	<0.001	27,941	<0.001	<0.001
Fat	2,516	14,142	<0.001	<0.001	0.004	14,193	<0.001	0.002
NumTrt								
HCW	2,525	23,957	<0.001	<0.001	0.002	24,613	<0.001	<0.001
LM area	2,518	17,552	<0.001	<0.001	0.0003	17,654	<0.001	<0.001
MS	2,524	27,865	0.872	<0.001	<0.001	27,928	<0.001	0.004
Fat	2,516	14,142	<0.001	<0.001	0.029	14,193	<0.001	0.015
Mean lung score					<			
HCW	2,482	23,516	<0.001	<0.001	0.001	24,188	<0.001	<0.001
LM area	2,475	17,269	<0.001	<0.001	0.407	17,374	<0.001	0.396
MS	2,481	27,366	<0.001	0.855	0.007	27,427	<0.001	0.006
Fat	2,474	13,905	<0.001	<0.001	0.056	13,960	<0.001	0.06
Lesions present								
HCW	2,482	23,559	<0.001	<0.001	0.151	24,229	<0.001	0.643
LM area	2,475	17,279	<0.001	<0.001	0.444	17,384	<0.001	0.3
MS	2,481	27,406	<0.001	0.795	0.198	27,469	<0.001	0.191
Fat	2,474	13,914	<0.001	<0.001	0.23	13,969	<0.001	0.354
Total BRD								
HCW	2,525	23,979	<0.001	<0.001	0.097	24,643	<0.001	0.283
LM area	2,518	17,575	<0.001	<0.001	0.786	17,683	<0.001	0.686
MS	2,524	27,884	<0.001	0.879	0.031	27,949	<0.001	0.028
Fat	2,516	14,148	<0.001	<0.001	0.034	14,199	<0.001	0.052

[1]HCW = hot carcass weight (kg), LM area = longissimus muscle area (cm^2), MS = marbling score, Fat = subcutaneous backfat thickness (mm)

[2]Trt = BRD incidence ,NumTrt = number of BRD treatments, Mean lung score = average of two lung lesion scores, Lesions present = binary (yes/no), Total BRD = binary (yes/no) for Trt or Lesions present, or both.

[3]Effects are significant at $P < 0.05$

Table 4.8. Full model (M1) least squares means (± SE) for bovine respiratory disease (BRD) classification[1] effect on carcass traits[2]

	Classification level						
	0	0.5	1	1.5	2	2.5	3
Trt							
MS	405.54 (1.68)	-	-11.31 (3.31)*	-	-	-	-
Fat	13.16 (0.11)	-	-0.62 (0.22)*	-	-	-	-
NumTrt							
HCW	356.37 (0.77)	-	-1.08 (1.61)	-	-4.10 (3.05)	-	-16.48 (4.49)*
LM area	82.69 (0.22)	-	0.27 (0.45)	-	1.00 (0.86)	-	-4.95 (1.26)*
MS	405.55 (0.0)	-	-11.48 (3.66)*	-	-14.72 (9.36)	-	-1.02 (119.77)
Fat	13.16 (0.11)	-	-0.57 (0.23)*	-	-0.91 (0.43)*	-	-0.70 (0.65)
Mean lung score							
HCW	346.98 (2.29)	13.37 (3.71)*	14.30 (2.91)*	10.40 (2.66)*	8.47 (2.60)*	8.74 (2.74)*	6.98 (2.64)*
MS	390.66 (5.04)	15.72 (8.13)*	23.81 (6.40)*	14.03 (5.83)*	13.78 (5.71)*	7.55 (6.02)	8.36 (5.71)
Total BRD							
MS	406.48 (2.40)	-	-6.12 (2.83)*	-	-	-	-
Fat	13.25 (0.16)	-	-0.39 (0.18)*	-	-	-	-

[1]Trt = BRD incidence ,NumTrt = number of BRD treatments, Mean lung score = average of two lung lesion scores, Lesions present = binary (yes/no), Total BRD = binary (yes/no) for Trt or Lesions present, or both.

[2]HCW = hot carcass weight (kg), LM area = longissimus muscle area (cm^2), MS = marbling score, Fat = subcutaneous backfat thickness (mm)

* LSMEAN is different from animals that have never been treated (0) $P<0.05$

Table 4.9. Reduced model (M2) least squares means (± SE) for bovine respiratory disease (BRD) classification[1] effect on carcass traits[2]

	Classification level						
	0	0.5	1	1.5	2	2.5	3
Trt							
HCW	356.99 (0.87)	-	-4.36 (1.71)*	-	-	-	-
MS	406.63 (1.68)	-	-11.34 (3.30)*	-	-	-	-
Fat	13.18	-	-0.68 (0.22)*	-	-	-	-
NumTrt							
HCW	357.01 (0.87)	-	-2.89 (1.82)	-	-4.37 (3.44)	-	-22.96 (5.06)*
LM area	82.74 (0.22)	-	0.11 (0.46)	-	0.98 (0.87)	-	-5.56 (1.28)*
MS	405.64 (1.68)	-	-11.52 (3.53)*	-	-14.75 (6.68)*	-	-1.04 (9.81)
Fat	13.18 (0.11)	-	-0.62 (0.23)*	-	-0.92 (0.44)*	-	-0.89 (0.64)
Mean lung score							
HCW	346.92 (2.60)	11.44 (4.20)*	15.07 (3.30)*	9.44 (3.02)*	9.77 (2.95)*	9.23 (3.11)*	6.77 (2.99)*
MS	390.70 (5.04)	15.57 (8.13)*	24.11 (6.38)*	14.04 (5.83)*	13.88 (5.70)*	7.51 (6.01)	8.33 (5.79)
Fat	12.37 (0.33)	0.85 (0.533)	1.04 (0.42)*	0.86 (0.38)*	0.78 (0.37)*	0.67 (0.39)	0.21 (0.38)
Total BRD							
MS	406.62 (2.40)	-	-6.19 (2.82)*				
Fat	13.23 (0.16)	-	- 0.36 (0.18)*				

[1]Trt = BRD incidence ,NumTrt = number of BRD treatments, Mean lung score = average of two lung lesion scores, Lesions present = binary (yes/no), Total BRD = binary (yes/no) for Trt or Lesions present, or both.
[2]HCW = hot carcass weight (kg), LM area = longissimus muscle area (cm^2), MS = marbling score, Fat = subcutaneous backfat thickness (mm)
* LSMEAN is different from animals that have never been treated (0) $P<0.05$

5 Estimation of genetic parameters for bovine respiratory disease and associated carcass traits in feedlot steers

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5.1 Abstract

ABSTRACT: Bovine respiratory disease is one of the most prevalent and economically burdening diseases facing the beef cattle industry. The economic impact of the disease makes it a primary candidate for research to improve health and profitability of beef cattle. Therefore, the primary objectives of this study were to estimate variance components (**BRD**) in feedlot cattle using feedlot treatment records (**Trt**), number of treatments (**NumTrt**), mean lung score, lesions present, and a combination of Trt and lung lesions present into total BRD. The second objective was to evaluate genetic correlations of the disease classifications with HCW, LM are, marbling score (**MS**), and subcutaneous backfat thickness (**Fat)** to identify any potential genetic antagonisms. Heritability estimates of BRD were 0.15 ± 0.06, 0.04 ± 0.03, 0.0 ± 0.0, 0.04 ± 0.06, and 0.07 ± 0.06 for Trt, NumTrt, mean lung score, lesions present, and total BRD, respectively. Genetic correlations were not estimated for mean lung score due to the lack of genetic variability (0.0 ± 0.0). Genetic correlations of Trt with carcass traits were unfavorable (0.19 ± 0.30) for HCW and LM area (0.03 ± 0.25), and favorable for MS (0.30 ± 0.21) and Fat (-0.004 ± .26). Genetic correlations for NumTrt were similar at 0.23 ± 0.42, -0.05 ± 0.35, -0.29 ± 0.29, and -0.06 ± 0.35, between HCW, LM area, MS, and Fat, respectively. Estimates of genetic correlations for presence of lesions were zero for all traits. Estimates for total BRD were opposite when compared to Trt, and NumTrt at -0.03 ± 0.4, -0.35 ± 0.36, 0.28 ±0.30, and 0.12 ± 0.35 between HCW, LM area, MS, and Fat, respectively. Results indicate that with selection genetic improvement can be made over time by utilizing feedlot health records. Genetic correlations between treatment

records and carcass traits were in general favorable and would increase profitability when incorporated into selection programs.

Key words: beef cattle, bovine respiratory disease, carcass trait, health

5.2 Introduction

Bovine respiratory disease is the single most costly disease facing the beef cattle industry (USDA NAHMS, 2000). Economic losses from postweaning incidence of BRD have been estimated to range from $13.90 to $92.26 per head for treated animals (McNeill et al., 1996; Faber et al., 1999; Snowder et al., 2006; Schneider et al., 2009). Griffin et al., (1997) estimated annual losses to the US cattle industry to near $1 billion, while $3 billion is spent on prevention and treatment costs.

As the pathology is further characterized for the causative agents, there is potential for treatment and prevention protocols to be developed to dramatically incidence of BRD. Though improvements in treatment and prevention protocols may reduce indirect costs associated with production loss, it would have marginal to no impact on cost already associated with treatment and prevention (McNeill et al., 1996; Faber et al., 1999; Snowder et al., 2006; Schneider et al., 2009). Consumers of the beef industry have become increasingly aware of animal well being, treatment, and the environmental conditions in which animals are raised. The consumer base also expects an increasing amount of red meat products to be free from antimicrobial and therapeutic drug residues used in the treatment of infected animals. Concern is also increasing over the overuse of

antibiotics in animal agriculture, and the implications that may have on the development of antibiotic resistant organisms in human medicine.

Selection for disease resistance is an alternative method to reduce economic losses and minimize public concern for animal welfare. Before selection schemes can be implemented it is important to understand the genetic variation within populations and any potential genetic antagonisms with other economically relevant traits (Golden et al., 2000). Therefore, the objectives of this study were to estimate direct genetic parameters for different classifications of BRD in feedlot steers and genetic and environmental correlations with HCW, LM area, marbling score (**MS**), and subcutaneous backfat thickness (**Fat**) for subsequent use in genetic evaluations.

5.3 Materials and Methods

All experimental procedures were reviewed and accepted by the Colorado State University Animal Care and Use Committee (07-230A-01). Crossbred steer calves, described previously in chapter 4.3, were shipped to a commercial feedlot in Southeastern Colorado in 2007 and 2008 (n = 1,551 and 1,319; respectively). Contemporary group was defined as combination of year, ranch unit of origin, and feedlot pen for disease classifications during the feeding period. An additional level of harvest date was included for disease classifications and carcass traits collected at harvest.

5.3.1 Disease Classification

Genetic parameters were estimated using several varying methods to classify disease occurrence. The trait of interest was defined to be susceptibility to BRD which was defined as an animal within the population with a higher risk of becoming affected with BRD and is less likely to moderate the microbial lifecycle and remain clinically healthy when exposed to causative agents. Numbers of available records for each classification group are given in Table 5.1. The first classification included binary treatment records during the feeding phase (**Trt**). Animals showing clinical signs of BRD and with subsequent treatment were coded as a 1 and untreated animals as 0. The number of treatments (**NumTrt**) an animal received, 0, 1,2 or 3, for animals receiving no, one, two, or three or more treatments during the feeding period. Mean lung score was used as an indicator of lung lesion severity, and was calculated as a mean of lung scores from two trained evaluators. A modified system described by Bryant et al., (1999) was implemented for the collection of lung lesion scores at commercial plant chain speed. The system scores lungs on a scale of 0 to 3 where a score of 0 indicated normal lung which may include small depressed healed areas; a score of 1 has lung damage in less than 5% of the total lung volume, minimal adhesions or pleuritis (fibrin tags); a score of 2 indicates numerous adhesions affecting more than one Anterior Ventral lobe, and a score of 3 had large amount of missing lung tissue caused by pleuritis and/or adhesions greater than 15% of the total lung volume, or active lymph nodes indicating a current infection. The binary classification of lung lesions present at harvest, where steers with moderate to severe lesions (mean lung score > 1.5) were classified as a 1 and steers with minimal lesions (mean lung score < 1.5) were classified as 0. The final classification combined

both Trt and presence of lesions into a binary variable (**total BRD**) to encompass steers that did not have lung scores due to, morbidity in the feedlot, premature culling due to chronic illness and lameness, and loss of identification at time of harvest. A steer was categorized as a 1 if they had an observed "1" for Trt during the feeding period, or a "1" for lesion present, or both. A steer assigned a "0" had was never treated and had no lesions present at harvest.

5.3.2 Pedigree information

Calves in the study were a result of multi-sire breeding pastures. Sire identification was performed through DNA sampling of both sire and progeny through a commercial lab. Eighty one percent of calves were successfully sire identified (n = 2,331). A historical pedigree was obtained from the ranch of origin. A five generation sire pedigree (n = 3,255) was constructed for the purpose of variance component estimation. The pedigree consisted of 386 sires. There were 309 sires with progeny with data. Of the sires with progeny with data, 270 (87%) had more than two progeny with a mean of 8 progeny per sire (SD = 6.9) and a minimum of 2 and maximum 60 progeny.

5.3.3 Variance Component Estimation

Single trait sire model was used to estimate direct genetic effects for the disease classification of Trt, NumTrt, mean lung lesion score, lesions present, and total BRD. The only fixed effect was contemporary group which was defined as ranch unit of origin, commercial feedlot pen, and year for Trt and NumTrt, with the addition of harvest date

for mean lung lesion score, lesion present, and total BRD. The random effect of sire was included to estimate additive genetic effects, which were assumed to be continuous for NumTrt and mean lung score. Due to the binary nature of Trt, lesions present and total BRD observations, a probit threshold link function was fitted to convert observations to the continuous underlying scale (Gianola and Foulley, 1983; Harville and Mee, 1984). The model used to estimate direct genetic effects is given below:

$$\mathbf{y} = \mathbf{Xb} + \mathbf{Zu} + \mathbf{e}$$

Where **y** is a vector of observations for each disease classification with pseudo observations on the underlying scale for Trt, lung lesions present, and total BRD. The **X** and **Z** are known incidence matrices for fixed and random effects, respectively; b is a vector of fixed effect solutions, u is a vector of unknown random direct genetic effects, and e is a vector of random error terms. The model moments were assumed to be,

$$E\begin{bmatrix} y \\ u \\ e \end{bmatrix} = \begin{bmatrix} Xb \\ 0 \\ 0 \end{bmatrix} \text{ and}$$

$$\text{var}\begin{bmatrix} u \\ e \end{bmatrix} = \begin{bmatrix} \frac{1}{4}\sigma_g^2 & 0 \\ 0 & \sigma_e^2 \end{bmatrix},$$

Where σ_g^2 and σ_e^2 are direct genetic and error variance, respectively.

A bivariate sire model was used to estimate (co)variance parameters between HCW and LM area and between MS and Fat. Lean traits were analyzed independently of fat traits. Summary statistics for evaluated carcass traits by Trt classification are reported in Table 5.2 A multivariate sire model was the used to estimate direct genetic and residual (co)variance parameters between disease traits that were found to have non zero heritabilities from the single trait evaluation and the suite of carcass traits. Disease classification was evaluated with lean traits (HCW and LM area) and fat traits (MS and Fat) separately. Fixed effects included contemporary group which was the same for disease traits as described earlier. The random effect of sire was included to estimate additive genetic effects, which were assumed to be continuous for all carcass traits, and a probit threshold link function was applied to relevant disease traits as earlier described, to convert binary observation to the underlying scale. The linear model used can be described in matrix notation as,

$$\begin{bmatrix}\mathbf{y_1}\\ \mathbf{y_2}\\ \mathbf{y_3}\end{bmatrix}=\begin{bmatrix}\mathbf{X_1} & \mathbf{0} & \mathbf{0}\\ \mathbf{0} & \mathbf{X_2} & \mathbf{0}\\ \mathbf{0} & \mathbf{0} & \mathbf{X_3}\end{bmatrix}\begin{bmatrix}\mathbf{b_1}\\ \mathbf{b_2}\\ \mathbf{b_3}\end{bmatrix}$$

$$+\begin{bmatrix}\mathbf{Z_1} & \mathbf{0} & \mathbf{0}\\ \mathbf{0} & \mathbf{Z_2} & \mathbf{0}\\ \mathbf{0} & \mathbf{0} & \mathbf{Z_3}\end{bmatrix}\begin{bmatrix}\mathbf{u_1}\\ \mathbf{u_2}\\ \mathbf{u_3}\end{bmatrix}+\begin{bmatrix}\mathbf{e_1}\\ \mathbf{e_2}\\ \mathbf{e_3}\end{bmatrix},$$

where known incidence matrices $\mathbf{X_i}$ and $\mathbf{Z_i}$ relate unknown fixed ($\mathbf{b_i}$), and direct genetic ($\mathbf{u_i}$) effects, respectively, to observations in $\mathbf{y_i}$ with subscripts 1, 2, and 3 denoting treatment classification k, and both HCW and LM area or MS and Fat. Observations for

binary disease traits are pseudo continuous observation on the underlying scale. $\mathbf{e_i}$ is vector a random residual terms specific to animals with records for trait i.

The first and second moments of the model were assumed to be

$$\mathbf{E}\begin{bmatrix}\mathbf{y}\\ \mathbf{u}\\ \mathbf{e}\end{bmatrix} = \begin{bmatrix}\mathbf{Xb}\\ \mathbf{0}\\ \mathbf{0}\end{bmatrix} \text{ and}$$

$$\mathbf{var}\begin{bmatrix}\mathbf{u}\\ \mathbf{e}\end{bmatrix} = \begin{bmatrix}\frac{\mathbf{1}}{\mathbf{4}}\mathbf{G_0} \otimes \mathbf{A} & \mathbf{0}\\ \mathbf{0} & \mathbf{R_n}\end{bmatrix}.$$

Where **u** and **e** are vectors of additive direct genetic and residual variance, respectively for each trait i. **A** is the Wright's numerator relationship matrix, ⊗ is the Kronecker product operator, $\mathbf{G_0}$ is the additive genetic (co)variance matrix and $\mathbf{R_n}$ is a matrix of residuals such that with only trait 1, trait 2, or trait 3 measured, $\sigma^2_{e_1}, \sigma^2_{e_2}, \sigma^2_{e_3}$ will be on the diagonal with subscripts defined above. With 2 traits measured, $\sigma^2_{e_i}$ will be on the diagonal and σ_{e_i,e_j} will be on the corresponding off-diagonal, where $\sigma^2_{e_i}$ is the variance due to residual effects for trait i, and σ_{e_i,e_j} is the residual covariance for i^{th} and j^{th} traits measured on the same animal with $i \neq j$. Due to mortality and loss of records, all traits were not measured on the same animal, and therefore some residual covariances are by definition zero such as feedlot treatment records with carcass traits for animals that died during the feeding period.

The genetic parameters for all traits and their standard errors were estimated using ASREML (Ver. 3.0, VSN International, Ltd., Hemel Hempstead, UK) which employs an average information REML algorithm. Additive genetic variance was calculated as 4 * sire variance estimated through ASREML.

5.4 Results and Discussion

5.4.1 Single trait evaluation

Genetic parameters for disease trait classification single trait analysis are presented in Table 5.3. Heritability for Trt on the underlying scale was estimated to be 0.15 ± 0.06. This estimate is higher than previous estimates of 0.07 reported by Schneider et al. (2009), and the observed estimates of ,0.04, 0.08 reported by Snowder et al. (2006 and 2007), respectively. However, when converted to the underlying scale the 0.08 estimate of Snowder et al., (2006) increased to 0.18. The current estimate is also larger that those reported for calves preweaning by Muggli-Crockett et al., (1992, 0.10 ± 0.02), Snowder et al., (2005, 0.07 ± 0.01 to 0.19 ± 0.01), and Schneider et al., (2009, 0.11 ± 0.06) (Table 5.4). The higher heritability estimate supports the findings proposed by Snowder et al., (2006) that heritability increases with increased disease prevalence. Prevalence rates used to estimate heritabilities of Schneider et al. (2009) and Snowder et al. (2006 and 2007) were reported at 9.43% and 17%, respectively, compared to the prevalence 27.6% in the current study. Similarly Bishop et al. (2010) proposed that estimates of heritability for disease resistance were underestimated due to prevalence, imperfect sensitivity and specificity, or the ability to accurately classify truly diseased animals. The current heritability estimate for BRD resistance indicates that with intensive

selection programs genetic progress can be made to improve resistance to BRD in the finishing phase. Analysis of NumTrt indicated a heritability to be 0.04 ± 0.03 which is similar to the 0.02 ± 0.03 estimate reported by Schneider et al. (2009) for number of treatments an animal received over the feeding period.

Lung lesions have been shown to be associated with ADG and some measures of carcass merit (Wittum et al., 1996; Bryant et al., 1999; Thompson et al., 2006; Schneider et al., 2009). However, to our knowledge heritability estimates have not been reported for lung lesion severity and presence. Evaluation of mean lung score which was calculated by averaging the lung score of two trained evaluators was estimated to be 0.0 ± 0.0. Similarly, analyses of lesions present revealed heritability on the underlying scale of 0.04 ± 0.06. These heritabilities are low and indicate that there is no to very little genetic variation associated with lung lesions at harvest. An evaluation of total BRD which incorporated both Trt and lung lesions present at harvest into a single binary classification revealed a slight increase in heritability to 0.07 ± 0.06.

5.4.2 Bivariate analysis of carcass data

Heritability, genetic covariance, and genetic correlations for bivariate analysis of lean and fat carcass traits are presented in Table 5.5. Heritabilities for all carcass traits were moderate ranging from 0.2 to 0.42. Heritability of HCW was estimated to be much lower than the 0.44 ± 0.08 reported by Snowder et al. (2007) and 0.71 ± 0.10 reported by Schneider et al. (2009). However, LM area and MS estimates were similar to those

reported by Snowder et al. (2007). Genetic covariances between HCW and LM area and MS and Fat were moderate and favorable at 0.34 ± 0.14 and 0.38 ± 0.13, respectively.

5.4.3 Genetic and environmental correlations

Genetic and environmental correlations were estimated for all methods of BRD characterization with all evaluated carcass traits, excluding mean lung score which was estimated to have no genetic variation (Table 5.6). The genetic correlation estimate for Trt and NumTrt with MS were -0.30 ± 0.21 and -0.29 ± 0.29, respectively. These estimates are in agreement with those reported by Schneider et al., (2009) who estimated genetic correlations between MS and Trt -0.43 ± 0.20 and MS and NumTrt -0.32 ± 0.37. Both estimates are higher than the 0.09 ± 0.13 reported by Snowder et al., (2007) between Trt and MS. Estimates for genetic correlations between Trt and NumTrt with HCW were unfavorable (Table 5.6) compared to the estimates reported by Schneider et al., (2009) of -0.22 ± 0.22 between Trt and HCW and -0.58 ± 0.45 between NumTrt and HCW. However, Snowder et al., (2007) reported a small but positive genetic correlation of 0.04 ± 0.14 between Trt and HCW. Genetic correlations for Trt and NumTrt with Fat were low and consistent with previous results (Snowder et al., 2007; Schneider et al.,2009). Lung lesions present was estimated to have no genetic correlation with any of the evaluated carcass traits in the current study. When evaluating genetic correlations of total BRD, there was a favorable estimate for LM area. However, estimates for MS and Fat with total BRD became unfavorable and opposite compared to Trt and NumTrt (Table 5.6). These estimates warrant further investigation as there is currently no literature reporting genetic parameters for lung lesions present at harvest.

Environmental correlations were near zero with low SE compared to genetic correlations (Table 5.6). These results in the current study would agree with the conclusion of Snowder et al. (2007), that environmental approaches including management and preventative therapies to decrease BRD incidence would improve overall carcass quality.

5.5 Implications

Heritability estimates in the current study for resistance to BRD were moderate and would suggest that with intensive selection programs genetic improvement could be made to reduce incidence of BRD during the finishing phase. It is important to take into consideration the genetic relationship between BRD incidence and carcass traits when developing successful breeding programs to improve the health of steers, while maintaining optimal economic outputs. This study illustrated favorable genetic correlations between both BRD incidence and number of treatments with both MS and Fat. Though standard errors were high, there was a genetic antagonism estimated between HCW and both BRD incidence and number of treatments. Further research is warranted to investigate the genetic correlations which were all estimated with high SE.

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Table 5.1. Bovine respiratory disease (BRD) prevalence and number of observations based on disease classification[1]

		Total Animals.	No. (%) Disease classification
Trt		2,870	
	0		2,078 (72.4)
	1		792 (27.6)
NumTrt		2,870	
	0		2,078 (72.4)
	1		539 (18.8)
	2		186 (6.5)
	3		67 (2.3)
Mean lung score		2,487	
	0		160 (6.4)
	$0 < i \leq 1$		381 (15.3)
	$1 < i \leq 2$		1,096 (44.1)
	$2 < i \leq 3$		850 (34.2)
Lesions present		2,487	
	0		1,026 (41.3)
	1		1,461 (58.7)
Total BRD		2,870	
	0		1,042 (36.3)
	1		1,828 (63.7)

[1] Trt = treatment (1=yes and 0=no, NumTrt = No. of respiratory treatments, Lesions present (1 – yes and 0 = no), Total BRD = Trt and Lung lesions present combined (1=yes and 0 = no)

Table 5.2. Summary statistics for evaluated carcass traits of treated and non-treated feedlot steers

	No. Records	Mean	SD	Minimum	Maximum
Treated					
HCW	632	355.7	35.0	172.8	454.1
LM area[1]	632	83.3	8.7	51.7	115.6
MS[2]	632	402.9	55.7	250.0	670.0
FAT[3]	632	12.6	4.1	0.8	33.5
Not Treated					
HCW	1893	353.9	32.2	154.7	474.0
LM area	1886	81.9	8.2	50.7	115.8
MS	1892	403.3	68.0	250.0	750.0
FAT	1884	13.2	4.2	1.0	36.6

[1]LM area = Loin muscle area (cm^2)
[2]MS = Marbling score
[3]Fat = subcutaneous fat thickness (mm)

Table 5.3. Variance component and heritability estimates for disease classification from single trait sire model analysis[1]

Classification[2,3]	σ^2_g	σ^2_e	σ^2_p	h^2
Trt	0.18 ± 0.09	1.0	1.18 ± 0.09	0.15 ± 0.06
NumTrt	0.014 ± 0.014	0.36 ± 0.01	0.37 ± 0.01	0.04 ± 0.03
Mean lung score	0.0 ± 0.0	0.64 ± 0.02	0.64 ± 0.02	0.0 ± 0.0
Lesions present	0.04 ± 0.07	1.0	1.04 ± 0.07	0.04 ± 0.06
Total BRD	0.07 ± 0.07	1.0	1.07 ± 0.06	0.07 ± 0.06

[1]σ^2_g = direct additive genetic variance, σ^2_e = environmental variance, $\sigma^2_{P <}$ phenotypic variance, and h^2 = heritability

[2]Trt = feedlot treatment records, NumTrt = number of feedlot treatments, Total BRD = animals with Trt or lesion present or both.

[3]Trt, Lesions present and Total BRD variances and heritabilities on the underlying scale with environmental variance = 1 by definition.

Table 5.4. Literature estimates of direct heritabilities on the observed scale for pre and postweaning incidence of BRD

	h^2_d[1]		
Breed	Preweaning	Postweaning	Source
Multi-Breed	0.10 ± 0.02	0.06 ± 0.07	Muggli-Cocket et al. (1992)
Multi-Breed	0.10 ± 0.01	-	Snowder et al. (2005)
Multi-Breed	-	0.08 ± 0.01	Snowder et al. (2006)
Multi-breed	-	0.08 ± 0.01	Snowder et al. (2007)
Angus Cross	0.12 ± 0.06	0.07 ± 0.04	Schneider et al. (2008)[2]

[1] h^2_d = direct heritability estimate
[2] estimates on the underlying scale

Table 5.5. Estimates of additive genetic variance and heritability (h^2 ± SE) for carcass traits[1] on the diagonal, genetic covariance ($\sigma_{g1,g2}$ ± SE)below, and genetic correlation (r_g ± SE) above the diagonal

	HCW	LM area	MS	Fat
HCW	314.9 0.24 ± 0.05	0.34 ± 0.14	-	-
LM area	31.01 ± 15.8	26.7 0.30 ± 0.05	-	-
MS	-	-	2445.2 0.42 ± 0.04	0.38 ± 0.13
Fat	-	-	36.97 ± 14.6	3.8 0.20 ± 0.04

[1]HCW = hot carcass weight (kg), LM area = Longissimus muscle area (cm^2), MS=marbling score, Fat = subcutaneous fat thickness (mm)

Table 5.6. Estimates of genetic ($r_g \pm SE$) and environmental ($r_e \pm SE$) correlations of bovine respiratory disease classifications[1] with carcass traits[2]

	Trt		NumTrt		Mean lung score		Total BRD	
	r_g	r_e	r_g	r_e	r_g	r_e	r_g	r_e
HCW	0.19 ± 0.30	-0.05 ± 0.02	0.23 ± 0.42	-0.07 ± 0.02	0.0 ± 0. 01	0.0 ± 0.02	-0.03 ± 0.40	-0.01 ± 0.02
LM area	0.03 ± 0.25	-0.01 ± 0.02	-0.05 ± 0.35	-0.02 ± 0.02	0.0 ± 0.01	0.01 ± 0.02	-0.35 ± 0.36	0.02 ± 0.02
MS	-0.30 ± 0.21	-0.06 ± 0.02	-0.29 ± 0.29	-0.03 ± 0.02	0.0 ± 0.01	-0.03 ± 0.02	0.28 ±0.30	-0.05 ± 0.02
Fat	-0.004 ± .26	-0.05 ± 0.02	-0.06 ± 0.35	-0.05 ± 0.02	0.0 ± 0.01	-0.02 ± 0.02	0.12 ± 0.35	-0.04 ± 0.02

[1] Trt = treatment yes/no, NumTrt = No. of respiratory treatments, Total BRD = Trt and Lesions present combined

[2] HCW = hot carcass weight (kg), LM area = longissimus muscle area (cm^2), MS = marbling score, Fat = subcutaneous fat thickness (mm)

Appendix

A. *R Code for Cluster Analysis*

```
## loading required library:
library(cluster)

## Set working directory:
setwd("C:/Documents and
Settings/Administrator/MyDocuments/Chase/Thesis/evaluation/cluster")

## Read in data file from current working directory:
dat1 <- read.table("data.fin", na.string=".",
        col.names=c("rfid", "vid", "year", "notch", "trt", "ntrt", "nother",
                        "ls1", "ls2", "mls", "lung", "brd", "group", "EADG", "ladg",
"adg"))

# 1 rfid        radio frequency identification number
# 2 vid         visual identification number
# 3 year        year recieved into feedlot
# 4 notch       ear notch previous ranch treatment (yes/no)
# 5 trt         binary response for brd treatment in feedlot
                1 = yes : 2 = no
# 6 ntrt        number of brd treatments recieved in feedlot
# 7 nother      number of other treatments animal recieved
# 8 ls1         lung score from tech one
# 9 ls2         lung scroe from tech two
# 10 mls        mean lung score
# 11 lung       binary response for mean lung scores
                (1 >= 1.5)=yes (2 < 1.5)=no
# 12 brd        total brd a combination of trt and or lung to make brd
                1=yes : 2 = no
# 13 group      group of chart comparing trt and lung
                        #       1 = no trt + no lung
                        #       2 = no trt + lung
                        #       3 = trt + no lung
                        #       4 = trt + lung
# 14 EADG       early adg
# 15 ladg       late adg
# 16 adg        overall ad
```

```
## Make new data for analysis. All fields must be non missing.
dat <- na.omit(dat1[,c(1,3,5,6,10,11,12,14,16)])

# 1 rfid        radio frequency identification number
# 2 year        year recieved into feedlot
# 3 trt         binary response for brd treatment in feedlot
                1 = yes : 2 = no
# 4 ntrt        number of brd treatments recieved in feedlot
# 5 mls         mean lung score
# 6 lung        binary response for mean lung scores (1 >= 1.5) (0 < 1.5)
# 7 brd         total brd a combination of trt and or lung to make brd
# 8 EADG        early adg
# 9 adg         overall ad

attach(dat)
dim(dat)
dat07 <- subset(dat, year==2007)
dat08 <- subset(dat, year==2008)

## Get prevalence for ntrt, lung and brd:
ntrt <- unlist(lapply(split(dat[,3],f=dat[,3]),length)); ntrt
        ntrt[1]/length(dat[,3]); ntrt[2]/length(dat[,3])

nlung <- unlist(lapply(split(dat[,6],f=dat[,6]),length)); nlung
        nlung[1]/length(dat[,6]); nlung[2]/length(dat[,6])

nbrd <- unlist(lapply(split(dat[,7],f=dat[,7]),length)); nbrd
        nbrd[1]/length(dat[,7]); nbrd[2]/length(dat[,7])


#########################################################################
##

## principal components and cluster of trt + mls + adg (Cluster 1)
pcs1 <- prcomp(dat[,c(3,5,9)], retex=T, scale=T)

clust.1 <- kmeans(dat[,c(3,5,9)],2) # 2 cluster solutions

aggregate(dat[,c(3,5,9)], by=list(clust.1$cluster), mean) ## get cluster means

nclust1 <- unlist(lapply(split(clust.1$clust,f=clust.1$clust),length)); nclust1
        nclust1[1]/length(clust.1$clust); nclust1[2]/length(clust.1$clust)


## principal components and cluster of trt + mls + EADG (Cluster 2)
pcs2 <- prcomp(dat[,c(3,5,8)], retex=T, scale=T)
```

```
clust.2 <- kmeans(dat[,c(3,5,8)],2) # 2 cluster solutions

aggregate(dat[,c(3,5,8)], by=list(clust.2$cluster), mean) ## get cluster means

nclust2 <- unlist(lapply(split(clust.2$clust,f=clust.2$clust),length)); nclust2
        nclust2[1]/length(clust.2$clust); nclust2[2]/length(clust.2$clust)

##########################################################################
###


write.table(cbind(dat[,c(3,6,7)], clust.1$clust,
clust.2$clust),file='dat.compare.txt',quote=FALSE,eol='\r',col.names=TRUE,row.names=
FALSE)
dat.compare <- read.table("compare.fin", header=T)
## compare TRT with LUNG
unlist(lapply(split(dat.compare[,1]==dat.compare[,2],f=dat.compare[,1]==dat.compare[,2]),length
))

## compare TRT with CLUST1
unlist(lapply(split(dat.compare[,1]==dat.compare[,4],f=dat.compare[,1]==dat.compare[,4]),length
))

## compare TRT with CLUST2
unlist(lapply(split(dat.compare[,1]==dat.compare[,5],f=dat.compare[,1]==dat.compare[,5]),length
))

## compare Lung with CLUST1
unlist(lapply(split(dat.compare[,2]==dat.compare[,4],f=dat.compare[,2]==dat.compare[,4]),length
))

## compare Lung with CLUST2
unlist(lapply(split(dat.compare[,2]==dat.compare[,5],f=dat.compare[,2]==dat.compare[,5]),length
))

## compare CLUST1 with CLUST2
unlist(lapply(split(dat.compare[,4]==dat.compare[,5],f=dat.compare[,4]==dat.compare[,5]),length
))

##########################################################################
###
```

```
## Plots of cluster groups
pdf("clusters.compare.pdf")
par(mfrow=c(2,2))

## Cluster 1 (TRT + MLS + ADG)
        clusplot(dat[,c(3,5,9)], dat.compare[,4], color=F, shade=F, lines=0,
                col.p=dat.compare[,4],
                main="Cluster grouping of BRD, lung score, and ADG")


## Cluster 2 (TRT + MLS + EADG)
        clusplot(dat[,c(3,5,8)], dat.compare[,5], color=F, shade=F, lines=0,
                col.p=dat.compare[,4],
                main="Cluster grouping of BRD, lung score, and EADG")

#############################################################################

###
##Make Kendall correlations
detach(dat)
attach(dat.compare)

cor(dat.compare)
cor.test(trt,clust.1.clust, method = "kendall")
cor.test(lung, clust.1.clust, method = "kendall")
cor.test(trt,clust.2.clust, method = "kendall")
cor.test(lung, clust.2.clust, method = "kendall")
cor.test(clust.1.clust, clust.2.clust, method = "kendall")
```

B. Example SAS Code for Regression Analysis

```
dm'output; clear; log; clear;' ;
run;

*## Import data file ;
proc import datafile='E:\thesis.dat.csv' out=dat dbms=csv replace;   getnames=yes;
run;

**** Check for proper import of all data;
proc contents data = dat;
run;

*** *Subset for for relevant data;
data d2;
        set dat;
        keep vid sire cg recweight trt ntrt lung meanls totalbrd hcg hage hcw rea ms fat gq
yg;
        fat = fat*10;
run;


**********Running mixed models with full data **********;
proc mixed data = d2;
        class vid hcg trt;
        model hcw = trt hcg / ddfm=kenwardroger solution;
        random vid;
        lsmeans trt / ADJUST=BON pdiff=control("0");

**********Running mixed models with full data plus receiving weight **********;
proc mixed data = d2;
        class vid hcg trt;
        model hcw = trt hcg / ddfm=kenwardroger solution;
        random vid;
        lsmeans trt / ADJUST=BON pdiff=control("0");
```

```
************* Subset data to contain only animals with harvest ages **************;
data d2sub;
        set d2;
        IF hage ^= .;
run;

proc means data=d2sub;
        var hage hcw rea ms fat;
run;

**********Running mixed models with subset data ******************;
proc mixed data = d2sub;
        class vid hcg trt;
        model hcw = trt hcg hage / ddfm=kenwardroger solution;
        random vid;
        lsmeans trt / ADJUST=BON pdiff=control("0");

*** Each model above is run with all carcass traits and all BRD classifications;
*** Carcass traits = Hot carcass weight, Loin muscle area, Marbling score, and
Subcutaneous fat
*** BRD classification = Trt, Ntrt, Mean lung score, Lesion present, and Total Brd
```

C. Example ASReml Code (trt.as)

```
Genetic Evaluation of BRD and carcass traits
        id              ## 1
        sire    !P      ## 2
        cg      !A*     ## 3
        trt             ## 4     Binary (1/0) = (yes/no)
        ntrt            ## 5
        mls             ## 6
        lung            ## 7     Binary (1/0) = (yes/no)
        brd             ## 8     Binary (1/0) = (yes/no)
        hcg     !A*     ##9
        hcw             ##10
        ms              ##11
        rea             ## 12
        fat             ## 13

../data/sped.stk        !MAKE       !ALPHA
../data/dat.run         !MAXIT 50   !MVINCLUDE      !ASUV       !DOPART
$1A

## Example of binary univariate BRD trait
!part 1
trt !bin !probit ~ cg !r sire
!end



## Example of continuous univariate BRD trait
!part 2
mls ~ hcg !r sire
!end
```

```
## Example of bivariate carcass analysis
!part 3
hcw rea ~ Trait Trait.hcg !r Trait.sire
1 2 1
0
Trait 0 US !GUUU
975.5
99.89   61.2

Trait.sire 2
2 0 US !GUUU
78.7
7.8      6.7
sire
!end

## Example of multivariate (binary BRD and continuous carcass analysis)
Trt hcw rea !bin !probit ~ Trait at(Tr,1).cg at(Tr,2).hcg at(Tr,3).hcg !r Trait.sire mv
1 2 1 !STEP 0.01
0
Trait 0 US !GFUUUUU
1
-1.8     1076
-0.06   125.4   65.9

Trait.sire 2
3 0 US !GUUUUUU
0.04
0.3      47.7
0.02     2.35     5.6
sire
!end
```

D. *EXAMPLE ASREML CODE (TRT.PIN)*

```
## Pin for univariate analysis

F       Rvar    2*1     #3

F       Gvar    1*4     #4

F       Pvar    3+4     #5

H       H2      4 5
```

```
## Pin for bivariate analysis

F       Rhcw            1*1     #7
F       RCOVhcwrea      2*1      #8
F       Rrea            3*1     #9

F       Ghcw            4*4     #10
F       GCOVhcwrea      5*4     #11
F       Grea            6*4     #12

F       Phcw            7+10     #13
F       Prea            9+12     #14

H       Hhcw            7 13
H       Hrea            9 14

R       RCORhcwrea      7 8 9

R       GCORhcwrea      10 11 12
```

Pin for multivariate analysis

F	Rtrt	3*1	#15
F	RCOVtrms	4*1	#16
F	Rms	5*1	#17
F	RCOVtrfat	6*1	#18
F	RCOVmsfat	7*1	#19
F	Rfat	8*1	#20
F	Gtrt	9*4	#21
F	GCOVtrms	10*4	#22
F	Gms	11*4	#23
F	GCOVtrfat	12*4	#24
F	GCOVmsfat	13*4	#25
F	Gfat	14*4	#26
F	Ptrt	15+21	#27
F	Pms	17+23	#28
F	Pfat	20+26	#29
H	Htrt	21 27	
H	Hms	23 28	
H	Hfat	26 29	
R	RCORtrms	15 16 17	
R	RCORtrfat	15 18 20	
R	RCORmsfat	17 19 20	
R	GCORtrms	21 22 23	
R	GCORtrfat	21 24 26	
R	GCORmsfat	23 24 25	