PHENOTYPIC AND GENETIC RELATIONSHIPS AMONG TEMPERAMENT, IMMUNE, AND CARCASS TRAITS IN BEEF CATTLE

by

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Abstract

Cattle temperament has historically influenced selection decisions due to ease of handling. However, temperament may also influence economically relevant traits. The purpose of this study was to investigate relationships between temperament, Bovine Respiratory Disease (BRD) incidence, and resulting carcass merit in feedlot steers. Across a two year period, 2,870 crossbred steers were shipped from a single ranch source to a feedlot. At the time of feedlot placement, as well as at the time of reimplantation, temperament was measured via chute score (CS) and exit velocity (EV). Blood samples were taken upon arrival to the feedlot to determine circulating concentrations of interleukin 8 (IL-8) and cortisol, both of which are involved in immune function. Performance traits, including weight and gains, were measured at feedlot placement (d 0), reimplantation (d 73-100), and again 59 to 70 days later. Recorded carcass data included HCW (HCW), USDA yield grade (YG), marbling score (MS), ribeye area, and lung scores. Phenotypic statistical analysis was performed with SAS statistical software (SAS Inst., Inc., Cary, NC) and genetic parameters were estimated using ASREML (Ver. 3.0, VSN International, Ltd., Hemel Hempstead, UK). The pedigree file included records of 7,177 animals with up to 7 generation of pedigree. Contemporary group (CG, n=11) included initial ranch unit, date of arrival to the feedlot, feedlot pen, and processing dates. Fixed effects included in the model were pre-feedlot entry BRD treatment and CG. Cattle with higher CS at placement subsequently had more BRD incidence ($P < 0.01$). There was a positive phenotypic correlation between placement CS and blood cortisol concentrations ($r = 0.07$; $P < 0.01$), and cattle with higher cortisol concentration contracted BRD more often than their calmer peers ($P < 0.05$). Circulating IL-8 concentration had no influence on feedlot health. At the time of reimplantation, cattle that had been treated for BRD in the feedlot had lower chute scores ($P < 0.001$). Heritability estimates for CS at placement, EV at placement, CS at reimplantation, and EV at reimplantation were 0.23, 0.17, 0.19, and 0.27, respectively. BRD incidence had a negative genetic correlation with all measures of temperament recorded at the second processing period.

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Chapter 1 - A Review of Literature

Introduction of Bovine Respiratory Disease

Bovine Respiratory Disease (BRD) is the most prevalent and costly disease affecting cattle in the U.S. feedlot sector (NAHMS, 2000). Bovine respiratory disease is a complex of diseases and is the result of an interaction between stress and naturally occurring viral and bacterial agents. Viral factors most commonly present in the development of BRD are infectious bovine rhinotracheitis (IBR), bovine viral diarrhea virus (BVDV), bovine respiratory syncytial virus (BRSV), and parainfluenza 3 (PI_3)(Salt et al., 2007). There are several additional bacterial agents involved, which include *Mannheimia haemolytica, Pasteurella multocida,* and *Haemophius somnus* (Ellis, 2001). A decrease in immune function in stress-inflicted cattle is generally the precursor to BRD. Stressors are predisposing factors and include weaning, relocation, and novel environments and social structures, which are commonly experienced by newly-received feedlot cattle (Snowder, 2009). Immunosuppressed cattle become candidates for infection from the previously stated agents. Cattle infected with BRD often exhibit signs of depression, decreased appetite, coughing, nasal and eye discharge, fever, and nasal congestion (Bagley, 1997). Elucidating genetic effects on disease susceptibility may indicate that there is potential to lessen its occurrence by selectively breeding against it.

Physiology of Bovine Respiratory Disease

Although BRD is generally associated with feedlot cattle, the disease complex can develop in cattle of any age. Typically, predisposing causative agents, such as immunological background, age, and stress, occur simultaneously and are complimentary to one another in the development of BRD (Callan and Garry, 2002; Duff and Galyean, 2007).

An interaction between stress, viral agents, and bacterial factors is nearly always involved in the development of this disease complex, and a list of common pathogens and stressors are shown on Table 1(Bagley, 1997). Cattle considered “healthy” commonly carry one or more of the pathogens in their upper respiratory tract, and with a well-functioning immune system, they are typically able to expel the agents via phagocytosis prior to manifestation and infection (Bagley, 1997). Stress, however, causes deviation from physiological homeostasis, and it hinders

the immune system by activating the hypothalamic-pituitary-adrenal axis (HPA) of the cattle (Lay and Wilson, 2001).

Stressors common to feedlot cattle, such as shipping and handling, activate the HPA (Lay and Wilson, 2001), and more temperamental cattle have a greater stress response as measured by cortisol (Stahringer et al., 1990; Curley et al., 2006). As a response to stressors, corticotropic releasing hormone (CRH) is released from the hypothalamus. After traveling through the hypothalamo-hypophyseal portal system, CRH acts on the anterior pituitary to cause the release of adrenocorticotropic hormone (ACTH). Adrenocorticotropic hormone circulates throughout the body and results in a release of glucocorticoids, namely cortisol, from the cortex of the adrenal gland. Such glucocorticoids break down protein, glycogen, and fat to increase the amount of circulating glucose (Lay and Wilson, 2001). An increase in circulating cortisol concentration also impairs the cell-mediated immunity of the animals by decreasing the number of macrophages, natural killer cells, T lymphoctyes, and cytokines (Jain et al., 1991; Pawlikowski, 1988). Cytokines, such as interleukin-8, assist in the cell-mediated immune response to bacterial infections, such as those found in the lung prefacing BRD development (Goubau et al., 2000). Following the release of cortisol and the subsequent suppressed immune function, viral agents proliferate and often migrate to the lower respiratory tract to cause infection, further inhibiting immune function (Martin and Bohac, 1986; Czuprynski et al., 2004).

Virulence of the causative pathogen(s) and the interaction between the pathogen and host determine whether the disease is expressed sub-clinically or clinically (Snowder, 2006a). Subclinical BRD shows no obvious or consistent signs for diagnosis, yet there is inflammation of the lungs due to pneumonia (Epperson, 1999). Clinical BRD, however, commonly shows signs of nasal and eye discharges, coughing, fever, decreased appetite, breathing difficulty, depression, and droopy ears (Bagley, 1997). In both clinical and subclinical cases of BRD, lung lesions are likely to develop.

Economic Impact of Bovine Respiratory Disease

Bovine respiratory disease costs the beef cattle industry an estimated $700 million annually (Griffin, 1997; NASS, 2006) and is responsible for approximately 75% of the morbidity and 50% of the mortalities experienced with feedlot cattle in the U.S. (Edwards, 1996; Smith, 1998). Six percent of the national mortality rate of all beef cattle is due to BRD (Snowder et al., 2006).

Bovine respiratory disease incurs several expenses, both directly and indirectly. Initially, costs accrue through practices that help prevent BRD incidence, such as vaccines. The beef industry spends approximately $3 billion on BRD prevention annually (Griffin, 1997). Upon contraction of BRD, costs become progressive. Snowder et al. (2006) estimated treatment to cost $15.57 on average per sick animal in the feedlot. Additional expenses are determined by the number of treatments, length of quarantine, and the effect of BRD on the ability of the animal to convert feed to lean meat.

Both subclinical and clinical cases of BRD decrease profits through decreased weight gains, veterinarian costs, and death. Total economic losses are dependent on the marketing objective set forth for the cattle. When cattle are sold on a live-weight basis, costs of disease are limited to death loss, treatment cost, decreased feed efficiency, decreased live weight (Larson, 2005), and extended days on feed. Cattle diagnosed with BRD during the finishing phase have significantly lower average daily gains than untreated cattle (McNeill et al, 1996; Gardner et al., 1999; Bateman et al., 1990). Gardner et al. (1999) found that steers treated for BRD had a mean of 7.5 kg lighter carcasses ($P < .01$) than untreated steers as a result of the lower average daily gains.

Marketing can also be done on a carcass merit basis through either a grid or branded product system. When cattle are sold based on carcass merit, profit is dependent on carcass weight, yield, and composition (Larson, 2005). A study performed by Gardner et al. (1999) showed that steers with lung lesions due to BRD infections yielded lighter HCW, lower dressing percentages, less internal fat, and lower marbling scores that those without lesions. Additionally, untreated steers yielded higher marbling scores that resulted in a higher percentage of carcasses graded U.S. Choice and U.S. Select (Gardner et al., 1999; Busby et al., 2009).

The health of cattle directly affects their feedlot performance and carcass quality (Baker, 2002). A better understanding of prevention and control methods, as well as behavioral and physiological factors that influence cattle health, is necessary to improve production efficiency and, ultimately, profitability.

Prevention and Control Measures for Bovine Respiratory Disease

Several measures have been taken in recent years to better prevent and control BRD incidence in the feedlot segment. Due to the ease of implementation, most of the recent efforts have been focused on improved management practices and pharmaceuticals (Bagley, 1997).

Because stress typically initiates immune suppression, management practices that abate environmental stress are most beneficial in decreasing herd sickness. Decreasing pathogen exposure and transmission through management practices will assist in decreasing disease incident. Due to the comingling nature of feedlots, this is a difficult task, and total eradication is not likely (Snowder et al., 2006b).

In addition to managing for less stressful environments, measures could be taken to increase cattle resistance to BRD through pharmaceuticals (Snowder et al., 2006b). This can be done through the use of vaccines and antibiotics. Vaccines, which can effectively reduce animal susceptibility to BRD, can reduce shedding of pathogens involved via bodily fluids. Less shedding helps to decrease transmission between calves. Although vaccination is effective and economical, only 28.4 percent of cow/calf producers vaccinate against the causative agents involved in BRD (USDA APHIS, 1997; USDA NAHMS, 2000a).

Preconditioning calves, a way of facilitating immunity against common BRD agents and minimizing the stress responses, is also beneficial in preventing BRD incidence (Cole, 1985; Speer, 2001; Dhuyvetter et al., 2005; Duff and Galyean, 2007). Preconditioning protocol varies by location, but typically includes weaning, vaccination, castration, and dehorning (Pritchard and Mendez, 1990). In a study performed by Roeber and Umberger (2002), cattle that were not preconditioned experienced a 41.6 percent greater incidence of BRD than their preconditioned peers. Preconditioning will not only assist in future health of the animal, but is economical for cow/calf producers (Dhuyvetter et al., 2005) as well as the finishing segment (Cravey, 1996; Roeber and Umberger, 2002). Dhuyvetter et al. (2005) estimated that producers can realize a $14.00 additional profit per preconditioned calf compared to the sale of calves that were not preconditioned when sold at weaning.

Antibiotics are effective in treating those infected with BRD, as well as helping to reduce shedding and decrease susceptibility in the future (Frank et al., 2000; Frank et al., 2002). USDA APHIS (2001) reported that 99.8 percent of all feedlots include injectable antibiotics in their BRD treatment regimen.

Although they are helpful for decreasing BRD incidence and treating those infected, antibiotics are costly and vary in efficacy between animals. In addition, the use of such pharmaceuticals increases skepticism among consumers regarding residues in the meat derived from injected animals. In order to reduce the disconnect and distrust between beef consumers and cattle producers, methods that are conducive to disease resistance without pharmaceuticals should take priority.

Genetic Selection for BRD Resistance

The combination of strong environmental infection pressures with inherently weak genetic resistance can result in contraction of infection and decreased performance in cattle (Van der Waaij et al., 2002). Decreased susceptibility could have the potential to improve cattle performance through decreasing the number of disease incidences. Although it is not likely that BRD will ever be completely eradicated, increased resistance through selective breeding could serve as a sustainable solution to BRD in the beef industry. By decreasing the genetic predisposition for disease, the potential for environmental insult to cause the development of disease lessens.

The initial challenge the beef industry is faced with is determining whether to select for disease resistance, tolerance, or increased immune function. Difficulties arise with each of these, as selection for each is dependent on correctly identifying their phenotypes followed by highly predictive genetic markers for the specified phenotypes (Snowder, 2006a). Additionally, cattle that appear to be unaffected by BRD may be thought to be disease resistant, which is a false assumption. Selection for immunity may lead to autoimmunity (Snowder, 2006a), and studies in swine indicate that selecting for immune responsiveness in one disease can have an antagonistic effect on the resistance to other diseases (Wilkie and Mallard, 1998). Determining whether all cattle are challenged equally, or given equal opportunity to develop BRD, is the initial difficulty when selecting for disease resistance. Furthermore, the magnitude to which signs of BRD are shown fluctuates between cattle, and infection may easily go undetected. A study performed by Wittum et al. (1996) depicts the discrepancy between visual diagnosis and true BRD incidence. In this study, 35 percent of 469 steers were treated for BRD between birth and slaughter. However, 72 percent of the 469 steers had lung lesions upon the time of slaughter.

Selection for disease resistance can be done both directly and indirectly. Direct selection includes observing animals in a specified environment for clinical expression of BRD, challenging breeding stock uniformly, and challenging relatives or clones of the breeding stock (Rothschild, 1998; Snowder, 2006a). As mentioned previously, simply observing animals for clinical expression amongst a contemporary group allows for much error, as not all individuals are exposed to the disease or are challenged to the same extent. This leads to a fallible assumption that all cattle that appear unaffected are disease resistant. Uniformly challenging breeding stock as well as relatives or clones can be costly, but will produce results with greater accuracy. Moreover, none of these methods account for immunological background. Indirect selection can be done by selecting for indicators for BRD resistance. Such indicators include biological and immunological responses of the host, as well as pathogen products (Snowder, 2006a). Accurate identification of similar indicator traits is necessary to facilitate a greater understanding of genetic interactions with BRD.

Breed differences in level of resistance have been elucidated in several studies, suggesting that genetics are influencing BRD resistance (Snowder et al., 2005; Snowder et al., 2006b; Muggli-Cockett et al., 1992). Snowder et al. (2005) collected birth and health records of 110,412 calves from the U.S. Meat Animal Research Center (MARC) from 1983 to 2002 (20 years). Cattle evaluated included Angus, Hereford, Red Poll, Charolais, Simmental, Limousin, Gelbvieh, Pinzgauer, Braunvieh, MARC I (composite), MARC II (composite), and MARC III (composite). In this study, purebred Braunvieh and MARC I (1/4 Braunvieh) had the highest incidences of BRD (18.85% and 16.67%, respectively), while Herefords had the lowest incidence (8.34%). Although Braunvieh had the highest incidence of BRD, they had the second lowest mortality rate. An earlier study conducted by Muggli-Cockett et al. (1992) had similar findings, where Braunvieh had the highest BRD frequency (20.1%) and Herefords had the lowest frequency (7%) of BRD. Due to genotype by environment interactions, the ability of a breed to resist disease pressures is influenced by its environment and is likely to fluctuate between environments (Snowder, 2006a).

Variation in response to disease challenges between breeds and individuals is largely due to genetics (Bishop et al., 2002; Nicholas, 2005). Muggli-Cockett et al. (1992) estimated the preweaning BRD incidence heritability to be 0.10 ± 0.02, while Snowder et al. (2006) estimated it to range from 0.00 to 0.26, depending on the breed. The heritability for disease resistance in

pre-weaned calves has been estimated at 0.26 (Schneider et al., 2009). When converted to an underlying continuous scale, however, this heritability increased to 0.48, suggesting that selection for disease resistance could have a greater response than originally expected (Schneider et al., 2009). Heritabilities are likely to be underestimated, as measurement error of disease detection and variability of expression of disease between individuals allows many infected animals to be unaccounted for.

Snowder et al. (2005) found large, negative correlations between direct and maternal effects, implying that females that appear genetically superior for BRD resistance produce progeny that are more susceptible to BRD. Additionally, results from this study showed that heterozygous calves (not purebreds) had significantly less incidences of BRD, suggesting that crossbreeding will decrease disease incidence.

Due to the complexity of the immune system and its interactions with other systems of the body, it is clear that disease resistance is polygenic in nature (Snowder, 2006a). Thus, determining which genes affect an animal's ability to ward off disease is difficult. By identifying a phenotype that defines disease resistance, there is potential for quantitative trait loci (QTL) involved in disease resistance to be discovered.

Although it could greatly improve animal health of the beef industry, expenses associated with research in genetic control of health and disease resistance have limited the number of studies. Moreover, researchers are challenged with providing an environment that challenges all animals equally with BRD. This is necessary to determine the common phenotypes of disease resistant cattle. If the phenotype is identified, investigation must be done to determine if it has an antagonistic effect on other economically relevant traits. If there are not antagonistic relationships, genetic selection for disease resistance is promising for the beef industry.

Temperament

The challenge with pathogen-associated diseases such as BRD is identifying indicator traits, available data, and DNA markers tests that allow researchers to genetically evaluate the cattle (Enns et al., 2011). Temperament influences the animal's stress response and has similar relationships with the HPA axis and immune function. This warrants further investigation to determine if temperament could serve as an accurate indicator trait of disease resistance or susceptibility.

Throughout history, researchers have acknowledged that cattle temperament plays a role in the efficiency and productivity of cattle (Elder et al. 1980a, 1980b; Hassall, 1974). However, little research was done due to the lack of known consistent measurements of cattle temperament. In 1982, Fordyce et al. developed a number of temperament tests, including the flight distance test, pen scores, and chute scores, to address this issue. Burrow et al. (1988) later developed the flight speed test, more commonly known as exit velocity, as a more effective assessment of cattle temperament than the flight distance test. As the new measurements began to appear, more physiological and performance associations with temperament were elucidated.

Currently, chute scores, pen scores, and exit velocities remain the most common measurements of animal temperament. Chute scores are assessed while the animal is in the squeeze chute (BIF, 2002). These are used to determine docility EPDs and are moderately heritable (BIF, 2002). Lower chute scores are preferred, as they denote a calmer, more docile animal. A pen score is taken while the cattle are in a 12 foot by 12 foot or 12 foot by 24 foot pen. To assess pen scores, two handlers enter the pen with a small group of cattle ($n \sim 5$). The handlers then slowly approach the cattle (Curley et al., 2006). The reaction of the cattle to the handlers' approach determines the animal's pen scores (BIF, 2002). Table 2 shows the guidelines for determining pen and chute scores. Exit velocity is the third measurement, and it is the velocity at which an animal leaves a restraining device, such as a squeeze chute (Burrow et al., 1988). Exit velocity can either be measured objectively in seconds using a photo electronic device or subjectively by visual appraisal using a six point categorical scale from 1 = slow to 6 = very fast. In using electronic equipment, the first timing trigger is often placed 6 feet beyond the headgate and the second timing trigger is often placed 12 feet from the headgate (6 feet between start and stop trigger) (BIF, 2002). Slower animals are less excited by the working chute, so lower EVs are preferable to producers. Petherek (2002) found that objective measures of temperament are highly repeatable and fluctuate little over time.

Positive correlations have been found between temperamental traits (catch scores, pen scores, and exit velocities) and cortisol levels in the blood (Curley et al., 2006; Cooke et al., 2009). Excitable cattle have higher concentrations of serum cortisol levels as a reaction to stressors and exhibit higher basal levels of cortisol (Curley et al, 2006; Curley et al., 2007). Although calm cattle have a greater initial response to stressors, temperamental cattle have significantly higher mean temperament responses at all points (Oliphint, 2006). Additionally,

Curley et al. (2007) found that temperamental animals sustain elevated cortisol levels for a longer duration and had greater pituitary and adrenal responses following a stressor than calm cattle. Such prolonged activation of the HPA and the consequent high levels of circulating serum cortisol has been shown to be detrimental to average daily gains and protein anabolism in cattle (Fell et al., 1999). Higher basal serum cortisol levels may suggest that easily excitable cattle are chronically stressed (Curley et al., 2007), resulting in a compromised immune response to disease pathogens, such as those necessary for BRD development.

Temperament differences occur across breeds, and *Bos indicus* cattle are more excitable than *Bos taurus* ($P < 0.01$; Voisinet et al., 1997). Similar correlations with breed type were found with BRD incidence (Muggli-Cockett et al., 1992; Snowder et al., 2005). Additionally, similar to those affected by BRD, temperamental cattle have lower average daily gains than their calmer peers (Voisinet et al., 1997; Grandin, 2003; Reinhardt et al., 2009), as well as lower HCW, marbling scores, dressing percentages, and yield grades (Larson, 2005). These relationships may suggest genetic linkage between genes involved in temperament development and disease susceptibility and may assist in genetic mapping. If there is linkage, temperament could serve as an inexpensive indicator trait of BRD susceptibility or resistance.

Reinhardt et al. (2009) conducted a study at the Tri-County Steer Carcass Futurity feedlots in Iowa examining the relationship between cattle temperament and BRD morbidity and mortality rates. A total of 13,540 steers were evaluated using the BIF docility assessments. Cattle given a 1 or 2 score were considered “docile”, those given a 3 or 4 were classified as “restless”, and those given a score of 5 or 6 were termed “aggressive”. Only 16.2 percent of the aggressive cattle were treated for sickness, whereas 19.2 percent of docile cattle were treated. In contrast, aggressive steers had a death loss of 1.91 percent and docile steers only had 1.09 percent. Higher mortality rates than morbidity rates may be due to a high number of cattle subclinically infected or not expressing common signs of clinical BRD, causing misdiagnosis (Reinhardt et al., 2009). This study indicated a relationship between temperament and disease incidence; however, the relationship was quadratic in nature (Reinhardt et al. 2009).

Oliphint et al. (2006) performed a similar study comparing cattle temperaments to immune function. In this study, exit velocities and cortisol concentrations were measured pre-, mid-, and post-shipment. Exit velocities were significantly different during post-shipment measurements than during pre-shipment measurements. Serum cortisol concentrations of calm

steers averaged 10.23 ± 1.52 ng/mL, while temperamental cattle had an average serum cortisol concentration of 15.35 ± 2.01 ng/mL over the evaluation period. Furthermore, serum cortisol concentration had a strong correlation with post-shipment exit velocity (r = 0 .68, P = 0.01) as well as midpoint exit velocity (r = 0.77, P = < 0.01). Thus, temperament appears to be an indicator of the magnitude of the animal's stress response. The magnitude of the stress response influences the susceptibility of that animal to disease.

Results from these studies suggest that there is an inter-relationship between temperament, stress, and disease incidence. Due to the novelty of cattle temperament research, few studies have been conducted to help reveal relationships linking the three. Literature suggests that temperament may affect disease susceptibility, however, and further research should be done to determine if they are influenced by the same genes.

Conclusions and Implications to Genetic Improvement of Beef Cattle

Bovine respiratory disease is highly detrimental to the economic value of the beef industry. Although pharmaceuticals are commercially available for assistance in prevention and treatment, genetic selection could prove more effective in decreasing transmission rates and yielding overall more productive cattle. Before genetic selection for disease resistance can occur, however, the disease resistant phenotype must be identified. This can be done if an inexpensive and humane way to equally challenge animals with BRD is developed. From there, response to future selection will be dependent on the proper characterization and accurate recording of BRD. Variability between and within breeds provides opportunity for breeders to base breeding decisions off of the potential selection differential.

Due to its influence on the stress response of an animal, temperament seems to be related to disease incidence. Determining both phenotypic and genetic relationships between temperament and animal health could assist in determining genes that influence both, allowing for selective breeding. Assuming there are no antagonistic relationships between resistance to disease and other economically relevant traits, selective breeding could prove economical in the long run to the beef industry. Additionally, reduced use of antibiotics could improve consumer acceptance of beef.

Figures and Tables

Table 1.1 Factors affecting the development of BRD.*

Stress Factors	**Viral Agents**	**Bacteria**
Heat	PI3	Pasteurella
Cold	IBR	Hemophilus
Dust	BVD	Other
Dampness	BRSV	
Injury	Adenovirus	
Fatigue	Rhinovirus	
Dehydration	Herpesvirus IV	
Hunger	Enterovirus	
Anxiety	MCF	
Irritant Gases	Reovirus	
Nutritional Deficiencies		
Surgery		

* Adapted from Bagley et al., 1997.

Table 1.2 Catch Score and Pen Score Measurements as termed by the BIF (2002).

Score	Temperament	Description
1	Docile	Mild disposition. Gentle and easily handled. Stands and moves slowly during processing. Undisturbed, settled, somewhat dull. Does not pull on headgate when in chute. Exits chute calmly.
2	Restless	Quieter than average, but may be stubborn during processing. May try to back out of chute or pull back on headgate. Some flicking of tail. Exits chute promptly.
3	Nervous	Typical temperament is manageable, but nervous and impatient. A moderate amount of struggling, movement and tail flicking. Repeated pushing and pulling on headgate. Exits chute briskly.
4	Flighty (Wild)	Jumpy and out of control, quivers and struggles violently. May bellow and froth at the mouth. Continuous tail flicking. Defecates and urinates during processing. Frantically runs fence line and may jump when penned individually. Exhibits long flight distance and exits chute wildly.
5	Aggressive	Aggressive. May be similar to Score 4, but with added aggressive behavior, fearfulness, extreme agitation, and continuous movement which may include jumping and bellowing while in chute. Exits chute frantically and may exhibit attack behavior when handled alone.
6	Very Aggressive	Very Aggressive. Extremely aggressive temperament. Thrashes about or attacks wildly when confined in small, tight places. Pronounced attack behavior.

References

Bagley, C. V. 1997. Bovine Respiratory Disease. Beef Cattle Handbook., Iowa Beef Center. Accessed Feb. 10, 2012. http://www.iowabeefcenter.org/Beef%20Cattle%20Handbook/Respiratory_Disease.pdf

Baker, M. 2002. Sick calves affect carcass quality, performance, and profitability. Beef Cattle Comments. Bulletin vol. 11.

Bateman, K. G., W. Martin, P. E. Shewen, and P. I. Menzies. 1990. An evaluation of antimicrobial therapy for undifferentiated bovine respiratory disease. Can. Vet. J. 31:689-696.

BIF. 2002. Beef Improvement Federation Guidelines: Behavioral Traits. Beef Improvement Federation. Accessed Sept. 23, 2011. http://www.beefimprovement.org/library/06guidelines.pdf

Bishop, S. C., J. Chesnais, M. J. Stear. 2002. Breeding for disease resistance: issues and opportunities. Proceedings of the 7th World Congress on Genetics Applied to Livestock Production. CDROM Communication No. 13-01.

Busby, D. 2009. Disposition-Convenience Trait or Economically Important? Iowa Beef Center. Lewis, IA. Accessed Sept. 24, 2011. http://www.iowabeefcenter.org/Docs_environment/Disposition.pdf

Callan, R.J. and F. B. Garry. 2002. Biosecurity and bovine respiratory disease. Vet. Clin. North Am Food An.18: 57-77.

Cole, R. K. 1968. Studies on genetic resistance to Marek disease. Avian Diseases 12:9-28.

Cooke, R. F., J. D. Arthington, D. B. Araujo, and G. C. Lamb. 2009. Effects of acclimation to human interaction on performance, temperament, physiological responses, and pregnancy rates of Brahman-crossbred cows. J. Anim. Sci. 87:4125-4132.

Cravey, M. D. 1996. Preconditioning Effect on Feedlot Performance. Page 33 in Southwest Nutri. Manag. Proc.

Curley, K. O., Jr., J. C. Paschal, T. H. Welsh, Jr., and R. D. Randel. 2006. Technical note: Exit velocity as a measure of cattle temperament is repeatable and associated with serum concentration of cortisol in Brahman bulls. J. Anim. Sci. 84:3100-3103.

Curley, K. O., Jr., D. A. Neuendorff, A. W. Lewis, J. J. Cleere, T. H. Welsh, Jr. and R. D. Randel. 2007. Functional characteristics of the bovine hypothalamic-pituitary-adrenal axis vary with temperament. Hormones and Behavior 53:20-27.

Czuprynski, C. J., F. Leite, M. Sylte, C. Kuckleburg, R. Schutlz, T. Inzana, E. Behling-Kelly and L. Corbeil. 2004. Complexities of the pathogenesis of *Mannheimia haemolytica* and *Haemophilus somnus* infections: challenges and potential opportunities for prevention? Animal Health Research Reviews. 5(2): 277-282.

Dhuyvetter, K. C., A. M. Bryant, and D. A. Blasi. 2005. Case Study: Preconditioning beef calves: Are expected premiums sufficient to justify the practice? Prof. Anim. Sci. 21:502-514.

Duff, G.S. and M.L. Galyean. 2007. Recent advances in management of highly stressed, newly received feedlot cattle. J. Anim. Sci. 85: 823-840.

Edwards, A. 1996. Respiratory disease of feedlot cattle in central USA. Bovine Pract. 30:5-7.

Ellis, J. A. 2001. The immunology of the bovine respiratory disease complex. Vet. Clin. North Am. Food An. 17:535-549.

Enns, R. M. 2011. Evidence of genetic variability in cattle health traits: Opportunities for improvement. Proceedings of the National Beef Improvement Federation. Bozeman, Montana.

Epperson, W. 1999. Lifetime Effects of Respiratory and Liver Disease on Cattle. Range Beef Cow Symposium. University of Nebraska-Lincoln. Lincoln, Nebraska.

Fell, L.R., I.G. Golditz, K.H. Walker, and D. L. Watson. 1999. Associations between temperament, performance, and immune function in cattle entering a commercial feedlot. Aust. J. Exp. Agric. 39: 795-802.

Fordyce, G., R. M. Dodt, and J. R. Wythes. 1988. Cattle temperaments in extensive beef herds in northern Queensland. 1. Factors affecting temperament. Aust. J. Exp. Agric. 28: 683-687.

Frank, G. H., R. E. Briggs, R. W. Loan, C. W. Purdy and E. S. Zehr. 2000. Effects of tilmicosin treatment on *Pasteurella haemolytica* organisms in nasal secretion specimens of calves with respiratory tract disease. Am. J. Vet. Res. 61:525-529.

Frank, G. H., R. E. Briggs, G. C. Duff, R. W. Loan and C. W. Purdy. 2002. Effects of vaccination before transit and administration of flofenicol at time of arrival in a feedlot

on the health of transported calves and detection of *Mannheimia haemolytica* in nasal secretions. Am. J. Vet. Res. 63: 251-256.

Gardner, B. A., H. G. Dolezal, L. K. Bryant, F. N. Owens, and R. A. Smith. 1999. Health of finishing steers: Effects on performance, carcass traits, and meat tenderness. J. Anim. Sci. 77: 3168-3175.

Goubau, S., D. W. Morck, and A. Buret. 2000. Tilmicosin does not inhibit interleukin-8 gene expression in the bovine lung experimentally infected with *Mannheimia (Pasteurella) haemolytica*. Can. J. Vet. Res. 64: 238-242.

Grandin, Temple. 2003. Assessment of Temperament in Cattle and Its Effect on Weight Gain and Meat Quality and Other Recent Research on Hairwhorls, Coat Color, Bone Thickness, and Fertility. Accessed Sept. 21, 2011 <http://www.grandin.com/behaviour/principles/assessment.temperament.html>.

Griffin, D. 1997. Economic impact associated with respiratory disease in beef cattle. Vet. Clin. North Am. Food An. 3: 367-377.

Jain, R., D. Zwickler, C. Hollander, H. Brand, A. Saperstein, B. Hutchinson, C. Brown and T. Audhya. 1991. CRF modulates the immune response to stress in the rat. Endocrinology. 128: 1329-1336.

Larson, R. L. 2005. Effect of cattle disease on carcass traits. J. Anim. Sci. 83:37-43.

Lay, D.C. and M. E. Wilson. 2001. Physiological indicators of stress in domestic livestock. Int. Anim. Agric. Food Sci. Conf. Indianapolis, IN. Accessed Jan. 20, 2012. http://infoservet.isch.edu.cu/Soporte/@/%28REVISTA%29%202001%20Symposia%20Journal%20of%20Animal%20Science%20Lay.pdf

Martin, S. W. and J. G. Bohac. 1986. The association between serological titers in infectious bovine rhinotracheitis virus, bovine virus diarrhea virus, parainfluenza-3 virus, respiratory syncytial virus and treatment for respiratory disease in Ontario feedlot calves. Can. J. Vet. Res. 50:351-358.

McNeill, J. W., J. C. Paschal, M. S. McNeill and W. W. Morgan. 1996. Effects of morbidity on performance and profitability of feedlot steers. J. Anim. Sci. 74(Suppl. 1): 135 (Abstr.).

Muggli-Cockett, N. E., L. V. Cundiff and K. E. Gregory. 1992. Genetic Analysis of bovine respiratory disease in beef calves during the first year of life. J. Anim. Sci. 70: 2013-2019.

NAHMS. 2000. Feedlot 1999 Part II: Baseline Reference of Feedlot Health and Health Management. USDA, APHIS, National Animal Health Monitoring System. Accessed Jan. 20, 2012. http://www.aphis.usda.gov/vs/ceah/ncahs/nnahms/feedlot/#feedlot99.

NASS (National Agricultural Statistics Service). 2006. Agricultural Statistics Board, United States Department of Agriculture. Cattle and Calf Death Losses.

Nicholas, F. W. 2005. Animal breeding and disease. Philosophical Transactions of the Royal Society (B) 360: 1529-1536.

Oliphint, R. A. 2006. Evaluation of the inter-relationships of temperament, stress responsiveness and immune function in beef calves. M.S. Thesis. Texas A &M University, College Station.

Pawlikowski , M., P. Zelakowski, K. Dohler, and H. Stepien. 1988. Effects of two neuropeptides somatoliberin and CRF on human lymphocyte natural killer activity. Brain Behav. Immun. 2: 50-56.

Petherick, J.C., R. G. Holroyd, V. J. Doogan and B. K. Venus. 2002. Productivity, carcass and meat quality of lot-fed *Bos indicus* cross steers grouped according to temperament. Australian Journal of Experimental Agriculture 42 (4) : 389-398.

Prichard, R. H. and J. K. Mendez. 1990. Effects of preconditioning on pre- and post-shipment performance of feeder calves. J. Anim. Sci. 68:28-34.

Reinhardt, C. D., W. D. Busby and L. R. Corah. 2009. Relationship of various incoming cattle traits with feedlot performance and carcass traits. J. Anim. Sci. 87: 3030-3042.

Roeber, D. L. and W. J. Umberger. 2002. The economic value of preconditioning programs in beef production systems. Presented at 2002 American Agricultural Economics Association Annual Meetings. http://agecon.lib.umn.edu. Accessed Feb. 20, 2012.

Rothschild, M. F. 1998. Selection for disease resistance in the pig. Natl. Swine Improv. Fed. Proc. Raleigh, North Carolina.

Salt, J.S., S. J. Therasagayam, A. Wiseman, and A. R. Peters. 2007. Efficacy of a quadrivalent vaccine against respiratory diseases caused by bhv-1, pi3v, bvdv, and bvsv in experimentally infected calves. Vet. J. 174: 616-626.

Schneider, M. J., R. G. Tait, Jr., W. D. Busby, and J. M. Reecy. 2008. An evaluation genetic components of bovine respiratory disease and its influence on production traits. M. S. Thesis. Iowa State Univ., Ames.

Smith, R. A. 1998. Impact of disease on feedlot performance: A review. J. Anim. Sci. 76: 272-274.

Snowder, G. D., L. D. Van Vleck, L. V. Cundiff and G. L. Bennett. 2005. Influence of breed, heterozygosity, and disease incidence on estimates of variance components of respiratory disease in preweaned beef calves. J. Anim. Sci. 83:1247-1261.

Snowder , G. D. 2006a. Genetic Selection for Disease Resistance: Challenges and Opportunities. Natl. Beef Improv. Fed. Proc. Choctaw, MS.

Snowder, G. D., L. D. Van Vleck, L.V. Cundiff, and G. L. Bennett. 2006b. Bovine respiratory disease in feedlot cattle: Environmental, genetic, and economic factors. J. Anim. Sci. 84:1999-2008.

Snowder, G. D. 2009. Genetics, environment and bovine respiratory disease. Animal Health Research Reviews. 10, 117 – 119.

Speer, N. C., C. Young, and D. Roeber. 2001. The importance of preventing bovine respiratory disease. Bovine Pract. 35: 189-196.

Stahringer, R. C., R. D. Randel and D. A. Neuendorf. 1990. Effects of naloxone and animal temperament on serum luteinizing-hormone and cortisol concentrations in seasonally anestrous Brahman heifers. Theriogenology 34: 393-406.

USDA APHIS. 1997. Beef '97 Part III: Reference of 1997 beef cow-calf production management and disease control. Centers for Epidemiology and Animal Health. http://www.aphis.usda.gov/animal_health/nahms/beefcowcalf/downloads/beef97/Beef97_dr_PartIII.pdf Accessed Feb. 27, 2012.

USDA APHIS. 2001. Treatment of respiratory disease in U.S. feedlots. Info sheet. APHIS Veterinary Services. www.aphis.usda.gov/vs/ceah/ncahs/nahms/feedlot/feedlot99/FD99treatresp.pdf . Accessed Feb. 27, 2012.

Van der Waaij, E. H., P. Bijma, S. C. Bishop and J. A. van Arendonk. Using genetic markers for disease resistance to improve production under constant infection pressure. J. Anim. Sci. 80: 322-329.

Voisinet, B. D., T. Grandin, J. D. Tatum, S. F. O'Connor and J. J. Struthers. 1997. Feedlot cattle with calm temperaments have higher average daily gains than cattle with excitable temperaments. J. Anim. Sci. 75: 892-896.

Wilkie, B. and B. Mallard. 1998. Multi-trait selection for immune response: A possible alternative strategy for enhanced livestock health and productivity. In: J. Wiseman (Ed.), Progress in Pig Science, Nottingham University Press, Nottingham, pp. 29-38.

Wittum, T. E., N. E. Woollen, L. J. Perino, and E. T. Littledike. 1996. Relationships among treatment for respiratory tract disease, pulmonary lesions evident at slaughter and rate of weight gain in feedlot cattle. J. Am. Vet. Med. Assoc. 209: 814-818.

Chapter 2 - Relationships among temperament, immune, and carcass traits in beef cattle

Introduction

Historically, cattle producers have selected for docile temperaments simply for management convenience because calmer animals are conducive to safe environments for their peers, as well as their handlers. As many producers would acknowledge, however, there seems to be a relationship between temperament and health, and calmer cattle tend to frequent the working chute for treatment of disease less often.

Positive correlations have been found in cattle between temperament traits (chute scores, pen scores, and chute exit velocities) and cortisol concentration in the blood, suggesting that more excitable cattle are easily stressed (Curley et al., 2006; Cooke et al., 2009). Additionally, Curley et al. (2007) found that easily excitable animals sustain elevated cortisol concentrations for a longer duration and had greater pituitary and adrenal responses following a stressor than calm cattle. Temperamental cattle have significantly higher mean temperament responses at all points (Oliphint, 2006). Higher basal serum cortisol concentrations may suggest that easily excitable cattle are chronically stressed (Curley et al., 2007), possibly resulting in a compromised immune response to disease pathogens, such as those necessary for Bovine Respiratory Disease (BRD) development.

Cattle diagnosed with BRD during the finishing phase have shown significantly lower average daily gains than untreated cattle (McNeill et al, 1996; Gardner et al., 1999; Bateman et al., 1990). Gardner et al. (1999) found that steers treated for BRD had a mean of 7.5 kg lighter carcasses ($P < .01$) than untreated steers as a result of the lower ADG. Gardner et al. (1999) also showed that steers with lung lesions due to BRD infections yield lighter HCW, lower dressing percentages, less internal fat, and lower marbling scores that those without lesions. Additionally, untreated steers yield higher marbling scores that result in a higher percentage of carcasses graded U.S. Choice and U.S. Select (Gardner et al., 1999).

This study was conducted to further investigate the relationships between cattle temperament (measured by chute score and exit velocity), immunological factors, and a range of economically relevant performance traits.

Materials and Methods

Animals

The Colorado State University Animal Care and Use Committee approved all experimental procedures (07-230A-01).

Crossbred steers were provided by a single ranch source with 3 units in western Nebraska in November of 2007 (n=1,551) and 2008 (n=1,319). Steers were shipped 536 km from their ranch unit to a commercial feedlot in southeastern Colorado. The first processing occurred 1 to 2 days after placement in the feedlot. Steers were placed in pens (6 pens in 2007, 5 pens in 2008), averaging 260 head per pen across the 2 years.

Processing

In Year 1 (2007), steers were given a radio frequency identification tag and a visual identification tag, then weighed at the time of feedlot placement. Ultrasound measures were taken at this time. Samples taken at initial processing included 30 mL of blood for circulating interleukin 8 (IL-8) and cortisol concentrations and a tissue sample from the ear to determine if the animal was infected with bovine viral diarrhea (BVD). Those that tested positive for a persistent infection of BVD were removed from the project. All animals were administered an oral parasiticide (Synanthic, Boehringer Ingelheim, St. Joseph, MO) a pour-on parasiticide (Promectin, Vedco, St. Joseph, MO), as well as a growth promotant (Revalor-IS, Merck, Summit, NJ). Vaccinations were not administered at the time of first processing in Year 1.

Initial processing for Year 2 (2008) calves included similar samples and ultrasound measures, with the addition of application of rumen temperature sensing boluses. Initial processing in Year 2 also included an injectable parasiticide (Noromectin, Norbrook Labs, Lenexa, KS). Due to the high rate of BRD contraction in the feedlot in Year 1 (45%) and subsequent expenses, 2 vaccinations, Pyramid 2 + Type II BVD and Presponse SQ (both from Boehringer Ingelheim, St. Joseph, MO) were administered in Year 2. Both vaccines prepare the immune system of the animal to respond to *Pasteurella multocida* and *Mannheimia haemolytica*, common viral and bacterial agents promoting the development of BRD. In Year 2, a 200-day delayed release growth promotant, Revalor-XS (Merck, Summit,NJ), was implanted rather than Revalor-IS.

Steers were processed at the time of reimplantation (73 to 100 days after initial processing) and again 59 to 70 days later. During the second and third processing of both years, measurements included ultrasound ribeye area, ultrasound back fat measured between the 12th and 13th rib, ultrasound intramuscular fat, and weight.

Growth calculations were determined by the difference between animal weight at feedlot reimplantation and weight at placement (GAIN1), as well as the difference between weights at the time of reimplantation and the third processing date (GAIN2). The two gain variables were summed to determine the total amount of gain in the feedlot.

Temperament Assessment

During processing at placement and reimplantation, each steer's temperament was assessed using exit velocity (EV; Burrow et al., 1988) and chute scores (CS; Grandin, 1993; BIF, 2002). Once restrained in the chute, cattle were assigned a subjective CS by two evaluators based on a 6-point scale defined by the Beef Improvement Federation (BIF) as follows: 1= docile, undisturbed, does not pull on headgate in chute; 2= restless, quiet, somewhat stubborn to move, some tail flicking; 3= nervous, moderately struggling to exit the chute, some movement and flicking of the tail; 4= flighty, frantically quivering and jumping, exhibiting continuous tail flicking, urinates and defecates in the chute; 5= aggressive, extremely agitated, continuous movement in the chute that includes jumping and bellowing; 6= pronounced attack behavior that is magnified in confined areas (BIF, 2002). The CS for each time point was averaged between the two evaluators. Because CS showed to be normally distributed, it was treated as a continuous variable for statistical analysis.

As cattle exited the chute, the flight time was measured over a distance of 1.83 m. Flight time has been defined as the time it takes an animal to cover a predetermined distance after leaving a confined area (Burrow et al., 1988). An EV, or the number of meters per second that the animal covered, was then determined by dividing the distance in meters by the recorded time in seconds. Temperament evaluators and others handling the cattle were positioned consistently behind the head catch to avoid influencing the response of the animal during both the EV and CS evaluations.

Harvest

Cattle were harvested between 68 and 97 days after the third processing (~d 225) at JBS Swift and Company plants in Dumas, TX and Greeley, CO in Year 1 and 2, respectively. Carcass data recorded included HCW, USDA yield grade, USDA quality grade (QG), numeric marbling scores (MS), ribeye area (REA), and lung scores. The calculated yield grade (YG) was derived from an assessment of percent kidney, pelvic, and heart fat, REA, HCW, and an adjusted fat thickness.

Two trained evaluators subjectively assigned lung scores of the aggregate lung post mortem. A lung score of 0 indicated a normal lung that may show healed areas. A lung score of 1 indicated lung damage in less than 5 percent of the total lung volume and minimal pleuritis (fibrin tags) or adhesions, while a lung score of 2 indicated damage from adhesions greater than 1 anterior ventral lobe. To be classified with a lung score of 3, cattle had to have a large amount of missing tissue caused by pleuritis, greater than 15 percent of the total lung volume damaged by adhesions, or active lymph nodes. The 2 evaluators' scores were then averaged for a single mean lung score. After determining that this variable was normally distributed, it was treated as a continuous variable for statistical analysis.

BRD Treatment

Cattle health was monitored by personnel of the commercial feedlot who rode through pens once daily to check for signs of illness. Steers were pulled for treatment based on the protocol set by the commercial feedlot. Steers that were considered ill were moved across the alley for evaluation by the Colorado State University South Eastern Colorado Research Center (SECRC) personnel. Steers exhibiting 2 or more signs of BRD, including lethargy, nasal and optical discharge, depression (determined by droopy ears), cough, and rectal temperatures greater than 40º C, were treated accordingly based on standard operating procedures set by SECRC. Steers diagnosed with BRD were monitored daily for 5 to 7 days post-treatment for the clinical signs, as well as weight and rectal temperature. Any steers still showing signs of infection between days 5 and 7 post-treatment, including a continuous rectal temperature above 40º C, were treated a second time. Those with rectal temperatures below 40º C between day 5 and 7 post-treatment were returned to their respective pens in the feedlot.

Metabolite and Cytokine Assays

Plasma was collected following centrifugation of whole blood samples and stored at -80º C until analysis. Both cortisol and IL-8 were measured using commercially available kits. More specifically, total plasma cortisol was measured using a commercially available radioimmunoassay kit, following manufacturer's protocol (Coat-A-Count; Diagnostic Products, Los Angeles, CA). Samples were analyzed in duplicate. A standard curve based on 0, 10, 50, 100, 200 and 500 µg/mL of cortisol was used to determine concentration of unknown samples. A high (200 µg/mL) and low (10 µg/mL) control were used to determine intra- and inter-assay coefficients of variability. A minimal detectable concentration was 2 ng/mL was used. Concentrations of IL-8 were measured using commercially available human ELISA kits according to the manufacturer's instructions, in which the antibody pairs have previously shown to cross-react with bovine IL-8 (Shuster et al., 1996, 1997; R&D Systems, Inc., Minneapolis, MN). Samples were analyzed in duplicate. A standard curve based on 0, 31.2, 62.5, 125, 250, 500, 1000, and 2000 pg/mL detected a minimal concentration of 3.5 pg/mL.

Statistical Analysis

Blood parameters, temperament measures, weights, and gains were analyzed using the general linear model procedure and a multivariate analysis of variance in SAS (SAS Inst., Inc., Cary, NC) with pre-feedlot BRD treatment, feedlot BRD treatment, and contemporary group (CG) as fixed effects. Contemporary group (n=11) included initial ranch unit, date of arrival to the feedlot, processing dates, and feedlot pen. Incidence of BRD pre- and post-entry to the feedlot was defined as binary traits. For each of these traits, 0 indicated no treatment, whereas a 1 indicated BRD treatment. Data from Year 1 and Year 2 were kept independent for analysis, then combined into a single dataset. Odds ratios were produced using the logistic regression procedure in SAS with mortality and feedlot BRD treatment included as response variables, while CG and pre-feedlot BRD treatment were treated as independent fixed effects.

Results and Discussion

Treatment for BRD prior to entry to the feedlot did not affect the likelihood of steer treatment or death in the feedlot in Year 1 (Table 2.1), Year 2 (Table 2.2), nor when the data from the 2 years was combined (Table 2.3). The combined dataset showed that 17.26 percent (n=53) of all steers that were treated for BRD during the preweaning period were treated for the

same complex after arrival to the feedlot, whereas 29.44 percent (n=755) of those without BRD prior to feedlot placement were later treated for the complex. Snowder et al. (2006) similarly reported that 13 percent of all calves treated for BRD during the preweaning period were later detected with BRD in the finishing stages. Bovine respiratory disease incidence in the feedlot significantly increased the probability of mortality in the feedlot by greater than 200 percent in each dataset (Table 2.1, 2.2, and 2.3).

BRD Treatment and Blood Parameters

Circulating serum cortisol concentration was negatively correlated with IL-8 concentration at the time of feedlot placement ($r = -0.07$, $P < 0.01$). Tobler et al. (1990) found that glucocorticoid hormones, such as cortisol, decreased expression of IL-8 and other cytokines in humans, which may explain a negative correlation between the 2 variables used here.

In Year 1, cattle treated for BRD prior to feedlot entry had a significantly lower circulating cortisol concentration during first processing ($P < 0.05$; Table 2.4), but not during Year 2 ($P > 0.05$; Table 2.5). When data from both years were combined, steers that had pre-entry treatment showed a significant decrease in cortisol concentrations at feedlot placement ($P < 0.05$; Table 2.6). This may suggest an acclimation effect, as the cattle had a lower physiological stress response to human interaction because it was not as novel of an environment to cattle that had already been treated. Curley et al. (2006) observed reductions in circulating cortisol concentrations over a period of 120 days that included a series of handling dates, and this decrease was attributed to habituation.

Similarly, cattle treated for BRD during their time in the feedlot had significantly greater cortisol concentrations at feedlot placement during Year 1 and in the combined dataset ($P < 0.05$; Tables 2.4 and 2.6), but not in Year 2 (Table 2.5). Buhman et al. (2000) showed that most cattle contract BRD in the first 27 days after feedlot placement. This time period is prior to the time of reimplantation. Thus, the decreased cortisol concentrations may be an acclimation effect as a result of 1 additional time in the working chute for BRD treatment. In Year 1 and when the data were combined, cattle treated for BRD in the feedlot had higher cortisol concentrations at initial processing than their non-treated peers ($P < 0.05$; Tables 2.4 and 2.6). This significance suggests that cattle that are more excitable are more likely to experience sickness as a result. Year 2 data contrasted the other 2 datasets and did not show a relationship between cortisol concentrations at

placement and post-feedlot entry BRD treatment (Table 2.5). The lack of significant relationships from the Year 2 dataset may be a result of low BRD treatment counts.

Bovine respiratory disease treatment prior to feedlot entry did not significantly influence circulating IL-8 concentration in either year separately ($P > 0.10$), nor when the yearly datasets were combined (Tables 2.4, 2.5, and 2.6). Cattle treated after feedlot entrance in Year 1, however, tended to have higher concentrations of circulating IL-8 than their peers ($P < 0.10$). Circulating IL-8 concentration did not influence BRD rates in the feedlot in Year 2 (Table 2.5) or when the data for the 2 years were combined (Table 2.6), but did tend to be associated with BRD rates in Year 1, as those with higher circulating IL-8 concentrations experienced more BRD incidence ($P < 0.1$; Table 2.4). Interleukin 8, a chemoattractive agent, is a cytokine that attracts neutrophils to a site to mediate tissue injury involved in disease (Caswell et. al, 1998). Thus, higher IL-8 concentrations for cattle that were later treated for BRD may have been due to a subclinical infection, which later developed into clinical BRD.

Table 2.7 shows the odds ratio estimates for IL-8 and cortisol with BRD treatment in the feedlot. A 1 pg/mL increase in IL-8 did not change the odds of BRD incidence in the feedlot. A 1 ng/mL increase in cortisol tended to slightly decrease the odds of BRD incidence in the feedlot (Table 2.7). Thus, as cortisol concentrations elevate in cattle, the odds of the animal requiring a treatment for BRD will generally decrease. An increase in cortisol concentration at the time of placement by 1 nanogram per milligram tended to slightly increase the odds of a 1 category increase in QG, whereas a change in IL-8 concentration showed no significant impact on QG (Table 2.8). This relationship between cortisol and QG contradicts previous literature, which suggested more temperamental cattle have a decreased MS, and thus a decreased QG (Voisinet et al., 1997). Increases in neither IL-8 nor cortisol concentration affected the odds of mortality (Table 2.9).

BRD Treatment and Temperament

Appraised CS at the time of feedlot placement was positively correlated with CS at the time of reimplantation, although the correlation was small (Table 2.10). Additionally, a weak positive correlation was found between CS at feedlot placement and both EV independently. This correlation with EV at both time points was also true for observed CS at the second processing. Grandin (1993) observed a similar perseverance of temperament observations in beef

cattle. Although there seems to be an effect of acclimation as shown by a decrease in circulating cortisol concentrations, that effect may be limited, providing some consistency in temperament.

Except for EV at the time of placement, all temperament measures were positively correlated with cortisol concentration (Table 2.11). Positive relationships between circulating cortisol concentrations and temperament have been reported previously, confirming that more excitable animals show significantly greater cortisol concentration than their calmer peers (Cooke et al., 2007; Curley et al., 2006; King et al., 2006, Stahringer et al., 1990). However, IL-8 concentration was not correlated with any of the temperament traits (Table 2.11).

In Year 1 and when the datasets were combined, average CS at first processing tended to be less for cattle that were treated prior to feedlot entry than their untreated peers ($P < 0.1$; Tables 2.4 and 2.6). The decrease in observed chute score may also be an effect of habituation, but this tendency was not consistent with Year 2 (Table 2.5). During the second processing, no difference in average CS was observed between steers treated and not treated prior to feedlot entry for all datasets. Average CS at both placement and reimplantation was significantly associated with BRD incidence in the feedlot for both years independently and when the datasets were combined (Tables 2.4, 2.5, and 2.6; $P < 0.05$). In all datasets, cattle that had a higher initial CS subsequently had more incidence of BRD (Table 2.4, 2.5, and 2.6; $P < 0.05$). At the time of reimplantation, however, cattle that were treated for BRD in the feedlot exhibited a lower CS (Table 2.4, 2.5, and 2.6; $P < 0.05$). Fell et al. (1999) experienced significantly more pulls from a pen of temperamental cattle in the feedlot than their calmer cohort. This study, however, included a total of only 24 head and only 1 of the steers pulled for treatment had BRD. Aside from this, no previous literature was found describing relationships between temperament and BRD incidence in the feedlot. Lung scores had no significant relationship with any of the temperament measures (Table 2.11).

During the first 2 processing periods, EV was not affected by treatment prior to feedlot entry for either year independently, nor when the data were combined (Tables 2.4, 2.5, and 2.6). Exit velocity at initial processing had no relationship with future BRD treatment. At the second processing, cattle treated in the feedlot in Year 1 had significantly lower EV than their untreated peers ($P = 0.0226$; Table 2.4). In Year 2, however, cattle that were treated in the feedlot had significantly higher EV at the second processing ($P = 0.0237$; Table 2.5). When the data were combined, there was no difference in exit velocities for cattle treated for BRD in the feedlot

versus their non-treated peers (Table 2.6). No previous literature supporting this result was found; however, the increase in temperament may be an effect of poor handling at the time of treatment. Thus, the result still supports an acclimation effect. Table 2.7 shows that a 1 m/s increase in EV at reimplantation increased the probability of BRD treatment in the feedlot by 17 percent, which was supported by the least squares mean in Table 2.5.

Results from the logistic regression procedure indicate that average CS and EV at the time of the second processing significantly influenced the likelihood of BRD incidence in the feedlot (Table 2.7). The initial analysis of risk of BRD incidence as a result of a 1 unit change in the average CS at the time of reimplantation included treatment prior to feedlot entry as a predictor variable. This variable was insignificant in the analysis ($P = .892$), and thus removed from the model to eliminate collinearity effects between pre-entry treatment and the CS at the second processing. The resulting odds ratio point estimate is 1.688. This suggests that with a 1 unit increase in CS at the time of reimplantation will result in a 68.8 percent increase in the probability of BRD incidence in the feedlot. As discussed previously, the least squares means of chute score for animals treated and not treated for BRD in the feedlot suggested that calmer cattle were the ones that contracted BRD. The conflictions of these 2 statistics can be explained by Figure 1, which shows a quadratic relationship between CS at reimplantation and BRD treatment in the feedlot. When CS at reimplantation was fit as a quadratic term in the model, it showed to be significant ($P < 0.01$). The final model produced fitted values that were 81.0 percent concordant with the observed treatment rates and 17.2 percent discordant. Thus, cattle that were on either end of the CS scale had a greater chance of experiencing BRD than their moderately temperamental peers. The quadratic effect of temperament on BRD rates was previously reported by Reinhardt et al. (2009), although the temperament measure was a subjective measurement of EV rather than CS. Odds of a change in QG or mortality were not affected by the temperament measurements, as shown by Table 2.8 and Table 2.9, respectively.

BRD Treatment, Temperament, and Performance

Steers that were previously treated for BRD were significantly heavier at the time of feedlot placement and reimplantation than their untreated peers in Year 2 ($P < 0.05$; Table 2.6). With the exception of the first 2 processing dates in Year 2, cattle weights at all 3 processing dates were no different for steers treated prior to the time of feedlot placement than for those not

treated Years 1 and 2 (Tables 2.4 and 2.5), nor when data from both years were combined (Table 2.6). Similarly, in Years 1 and 2 independently (Tables 2.4 and 2.5), as well as when the data were combined (Table 2.6), treatment prior to feedlot placement did not influence weight gains during the finishing stages.

For both years, as well as when the data were combined, there was no relationship found between weight at placement and treatment after feedlot entry (Tables 2.4, 2.5, and 2.6). This is consistent with findings from Martin et al. (1990), which showed that weight at time of placement did not prove to be significant in predicting BRD morbidity rates in the feedlot. However, steers that were treated in the feedlot had lighter ($P < 0.05$) body weights at the time of the second and third processing compared to those that were not treated in the feedlot in Years 1 and 2, as well as for both years combined (Tables 2.4, 2.5, and 2.6). Because BRD incidence commonly occurs within the first 27 days of feedlot placement (Buhman et al., 2000), the relatively lower body weights at later processings may be a direct result of poor health. All 3 datasets showed that GAIN1 was less ($P < 0.01$) for cattle treated for BRD during their time in the feedlot (Tables 2.4, 2.5, and 2.6). In Year 2 and when data from both years were combined, GAIN2 was significantly less ($P < 0.05$) for cattle treated for BRD in the feedlot as well (Tables 2.5 and 2.6). For Year 1, however, GAIN2 was not affected by post-feedlot entry treatment (Table 2.4). Total gain was significantly less ($P < 0.05$) in all 3 datasets for cattle treated while in the feedlot (Tables 2.4, 2.5, and 2.6). Cattle often exhibit a decreased appetite as a sign of BRD, resulting in lower gains (Schneider et al., 2009).

Exit velocity at the time of reimplantation was slightly negatively correlated with steer weights at the second and third processing (Table 2.11). Weaber and Creason (2010) found significant negative correlations between repeated measures of EV and repeated measures of weight, and reported that the average initial weight were associated with greater EV measures upon entrance to a post-weaning growth period. There was a correlation between GAIN1 and GAIN2 ($r = 0.2131$; $P < 0.001$), suggesting that those steers that gained most between the first and second processing were also the ones that gained the most between the second and third processing. Exit velocity and CS, each at the time of reimplantation, were negatively correlated with GAIN1, GAIN2, and the total gain in the feedlot (Table 2.11). This is consistent with the findings of Café et al. (2011) who showed that more excitable cattle gain less in the finishing stages than their less excitable peers.

Exit velocity observed at the second processing was negatively correlated with the HCW and YG, although the correlation is weak (Table 2.11). This suggests that cattle that were more flighty during the second processing exhibited smaller HCW and lower YG. Marbling, as measured by MS, was positively correlated with HCW (r = 0.1747; $P < 0.001$) and YG (r = 0.3084; $P < 0.001$), so those with heavier HCW and higher YG (often due to an increase in carcass fat) resulted in cuts with greater marbling percentages.

Café et al. (2011) found negative relationships between temperament measures and HCW. Table 2.11 shows that the only temperament measure that had a significant relationship with HCW was EV at the second processing. A weak negative correlation was found between MS and CS at placement (Table 2.11); however, MS showed no relationship with other temperament measures. There was no effect of steer temperament with postmortem lung score. Similar results were reported by Reinhardt et al. (2009), which studied effects of temperament on carcass characteristics in both heifers and steers. Neither sex displayed a relationship between temperament and subsequent lung score (Reinhardt et al., 2009). However, more excitable cattle did show a reduced MS than their calmer cohorts (Reinhardt et al., 2009). Decreased MS are to be expected with more flighty animals, as they have shown to have consistently greater concentrations of serum cortisol (Curley et al., 2007), which instigates uptake of glucose stored in the body.

Temperament Change

Table 2.12 shows the least squares means for the change in average CS from placement to reimplantation. Steers that began with an average CS of 2 or below increased significantly between the first and second processing ($P < 0.05$). The amount of temperament increase was greatest for those that began with the lowest chute score. Steers that began with an average chute score of 2.5 or above significantly decreased between the first and second processing. The amount of decrease was greatest for those that were observed with the highest initial CS. The changes in both calm cattle and excitable cattle are to be expected and may be an artifact of the CS appraisal scale, as the only change possible for those with a lower CS is an increase and the only change possible for steers with a higher CS is a decrease.

Over the course of feedlot placement through reimplantation, the mean EV increased from 2.971 ± 0.014 m/s to 3.032 ± 0.018 m/s, contradicting previous literature. A decrease in EV

has been previously observed; Brahman bulls have shown to have exit velocities that decrease over a period of 120 days in a ranch environment (Curley et al., 2006). Such observations in both studies could be attributed to acclimation; a decrease in EV results from positive previous experiences with the working chute and an increase in EV results from negative previous experiences with the working chute. Table 2.13 shows the number of cattle that transitioned in CS appraisal between the first and second processing. Most cattle either start or transition to a CS between 2 and 3.5.

The number of steers from each initial level of CS and their final observed CS from the second processing is shown on Table 2.14. Cattle with a beginning CS of 3 showed to be most consistent in CS with a total of 29.7 percent remaining in the same category.

Conclusions

Results suggest that temperament measures (i.e. CS and EV) were indicative of circulating cortisol concentrations. Easily excitable temperaments and subsequent high cortisol concentrations are associated with disease rates in the feedlot. These greater rates of BRD led to decreased carcass merits in the cattle. Thus, based off evidence from this study and previous literature, calmer cattle generally perform better and may be more economical as a result.

Figures and Tables

Figure 2.1 Histogram of Bovine Respiratory Disease (BRD) treatment rates in the feedlot by average chute score (CS) at the time of reimplantation

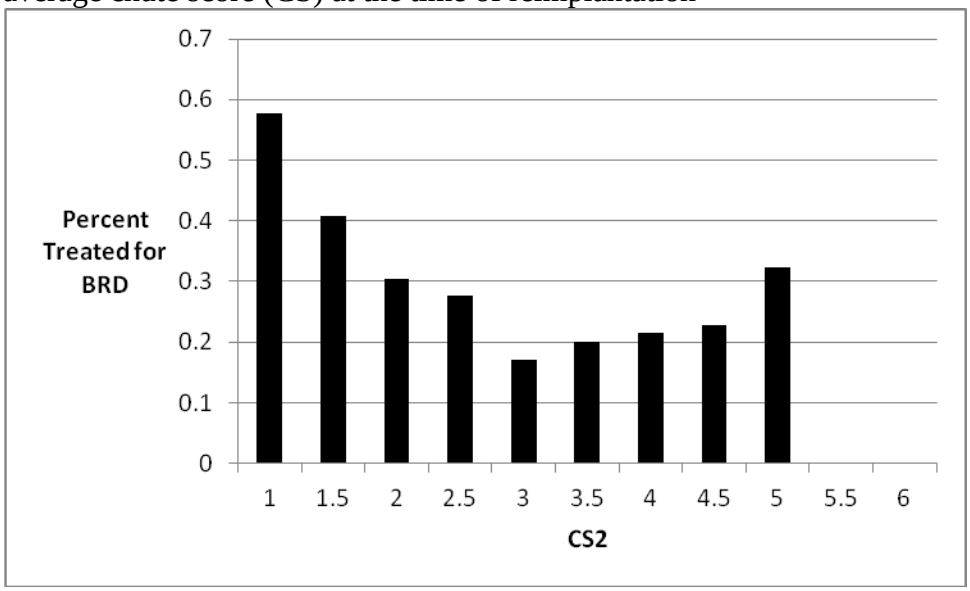

Table 2.1 Odds ratio estimates with associated 95% confidence intervals and P-values for the effect of treatment for bovine respiratory disease (BRD) before entry to the feedlot on post-entry BRD incidence and mortality, and the effect of post-entry BRD treatment on mortality for Year 1

Pre-entry Treatment			
Trait	**Odds Ratio Estimate**	**95% Confidence Interval**	**P-value**
Post-entry Treatment	1.073	(0.726, 1.586)	0.7220
Mortality	0.873	(0.361, 2.113)	0.7634
Post-entry Treatment			
Mortality	3.539	(2.190, 5.719)	< 0.0001

Table 2.2 Odds ratio estimates with associated 95% confidence intervals and P-values for the effect of treatment for bovine respiratory disease (BRD) before entry to the feedlot on post-entry BRD incidence and mortality, and the effect of post-entry BRD treatment on mortality for Year 2.

Pre-entry Treatment			
Trait	**Odds Ratio Estimate**	**95% Confidence Interval**	**P-Value**
Post-entry treatment	0.811	(0.173, 3.810)	0.7904
Mortality	2.916	(0.245, 34.708)	0.3971
Post-entry Treatment			
Mortality	8.734	(4.018, 18.982)	< 0.0001

Table 2.3 Odds ratio estimates with associated 95% confidence intervals and P-values for the effect of treatment for bovine respiratory disease (BRD) before entry to the feedlot on post-entry BRD incidence and mortality, and the effect of post-entry BRD treatment on mortality for Years 1 and 2 combined

Pre-entry Treatment			
Trait	**Odds Ratio Estimate**	**95% Confidence Interval**	**P-Value**
Post-entry treatment	1.056	(0.722, 1.542)	0.7796
Mortality	0.202	(0.455, 2.219)	0.9901
Post-entry Treatment			
Mortality	4.517	(2.931, 6.961)	< 0.0001

Table 2.4 Year 1 least squares means (±SE) for metabolic, temperament, and growth traits for steers that were treated or not for bovine respiratory disease (BRD) prior to feedlot entry and steers that were treated or not for BRD after feedlot entry.

Trait	Pre-entry			Post-entry		
	Treated	Not treated	P-value	Treated	Not treated	P-value
First Processing:						
IL-8 (pg/mL)	440.95	430.35	0.6450	447.05	424.25	0.0979
	(22.19)	(7.25)		(13.99)	(13.40)	
Cortisol (ng/mL)	25.33	28.34	0.0135	27.66	26.00	0.0232
	(1.18)	(0.39)		(0.74)	(0.71)	
Average chute score	2.83	2.97	0.0620	2.96	2.84	0.0097
	(0.08)	(0.02)		(0.05)	(0.05)	
Exit velocity (m/s)	2.90	2.96	0.4580	2.95	2.91	0.3172
	(0.06)	(0.02)		(0.04)	(0.04)	
Weight1 (kg)	224.36	226.20	0.3610	224.68	225.89	0.3160
	(1.94)	(0.63)		(1.22)	(1.17)	
Second Processing:						
Average Chute Score	2.51	2.50	0.8952	2.31	2.70	< 0.0001
	(0.08)	(0.03)		(0.05)	(0.05)	
Exit Velocity (m/s)	3.22	3.13	0.2796	3.12	3.24	0.0226
	(0.09)	(0.03)		(0.05)	(0.05)	
Weight2 (kg)	330.99	333.78	0.3712	328.04	336.73	< 0.0001
	(3.01)	(0.98)		(1.90)	(1.82)	
Third Processing:						
Weight3 (kg)	431.57	435.19	0.8665	101.18	100.16	0.4340
	(3.83)	(1.25)		(1.35)	(1.28)	

Gain2 (kg)	100.86	100.49	0.8665	101.18	100.16	0.4340
	(2.13)	(0.69)		(1.35)	(1.28)	
Total Gain (kg)	208.48	209.05	0.8584	206.18	211.35	0.0072
	(3.13)	(1.02)		(1.98)	(1.88)	

Table 2.5 Year 2 least squares means (±SE) for metabolic, temperament, and growth traits for steers that were treated and not treated prior to feedlot entry treatment prior to feedlot entry and steers that were treated and not treated after feedlot entry treatment.

	Pre-entry			Post-entry		
Trait	**Treated**	**Not treated**	**P-value**	**Treated**	**Not Treated**	**P-Value**
First Processing:						
IL-8 (pg/mL)	528.99	485.74	0.2707	513.30	501.43	0.6685
	(35.77)	(15.23)		(29.86)	(15.25)	
Cortisol (ng/mL)	42.40	45.18	0.2050	44.56	43.0163	0.3183
	(2.00)	(0.85)		(1.67)	(0.85)	
Average Chute Score	2.66	2.67	0.9285	2.73	2.60	0.0582
	(0.09)	(0.04)		(0.08)	(0.04)	
Exit Velocity (m/s)	2.97	2.97	0.9345	2.92	3.02	0.1984
	(0.09)	(0.014)		(0.08)	(0.04)	
Weight1 (kg)	225.92	215.49 (1.69)	0.0171	220.48	220.93	0.8830
	(3.98)			(3.32)	(1.70)	
Second Processing:						
Average Chute Score	2.44	2.42	0.8987	2.29	2.57	0.0001
	(0.09)	(0.04)		(0.08)	(0.04)	
Exit Velocity (m/s)	2.93	2.96	0.8215	3.03	2.86	0.0237
	(0.09)	(0.04)		(0.08)	(0.04)	
Third processing:						
Weight3 (kg)	494.43	482.00	0.1260	473.67	502.76	< 0.0001
	(7.46)	(3.25)		(6.41)	(3.17)	

Gain2 (kg)	105.95	107.90	0.6599	103.31	110.54	0.0274
	(4.06)	(1.78)		(3.51)	(1.72)	
Total Gain (kg)	271.19	269.12	0.7159	258.22	282.08	< 0.0001
	(5.24)	(2.30)		(4.52)	(2.22)	

Table 2.6 Least squares means (±SE) for metabolic, temperament, and growth for steers that were treated and not treated prior to feedlot entry and treated or not treated after feedlot entry for Years 1 and 2 combined.

Trait	Pre-entry			Post-entry		
	Treated	**Not Treated**	**P-value**	**Treated**	**Not Treated**	**P-value**
First Processing:						
IL-8 (pg/mL)	477.75	458.15	0.3283	477.69	458.20	0.1174
	(18.56)	(6.21)		(12.79)	(9.79)	
Cortisol (ng/mL)	33.09	36.04	0.0069	35.43	33.69	0.0105
	(1.01)	(0.34)		(0.70)	(0.53)	
Average Chute Score	2.73	2.84	0.0780	2.85	2.72	0.0012
	(0.06)	(0.02)		(0.04)	(0.03)	
Exit Velocity (m/s)	2.95	2.99	0.4269	2.98	2.95	0.4578
	(0.05)	(0.02)		(0.04)	(0.03)	
Weight1 (kg)	223.18	221.79	0.4890	222.12	222.86	0.5491
	(1.86)	(0.62)		(1.28)	(0.98)	
Second Processing:						
Average Chute Score	2.46	2.45	0.9070	2.27	2.64	< 0.0001
	(0.06)	(0.02)		(2.64)	(0.03)	
Exit Velocity (m/s)	3.06	3.00	0.3656	3.07	3.00	0.1409
	(0.06)	(0.02)		(0.04)	(0.03)	
Weight2 (kg)	357.69	355.61	0.4717	351.26	362.04	< 0.0001
	(2.66)	(0.89)		(1.84)	(1.41)	
Gain1 (kg)	133.81	133.69	0.9580	128.98	138.53	< 0.0001
	(2.10)	(0.70)		(1.45)	(1.11)	

Third processing:

Weight3 (kg)	461.39	460.34	0.7860	454.87	466.85	< 0.0001
	(3.57)	(1.20)		(2.47)	(1.88)	
Gain2 (kg)	104.70	105.04	0.8694	104.46	105.28	0.5357
	(1.95)	(0.66)		(1.35)	(1.03)	
Total Gain (kg)	239.54	239.25	0.9189	234.76	244.03	< 0.0001
	(2.68)	(2.68)		(1.86)	(1.42)	

Table 2.7 Odds ratio estimates with associated 95% confidence interval and P-values for exit velocity (m/s) and chute score at first and second processing (EV1, EV2, CS1, CS2), and circulating interleukin 8 (IL-8, pg/mL) and cortisol (CORT, ng/mL) concentrations at first processing with bovine respiratory disease treatment in the feedlot for Years 1 and 2 combined.

	Post-entry Treatment		
Trait	**Odds Ratio Estimate**	**95% Confidence Interval**	**P-value**
EV1	1.088	(0.961, 1.231)	0.1825
EV2	1.166	(1.055, 1.289)	0.0026
CS1	0.990	(0.882, 1.111)	0.8605
CS2	1.688	(1.501, 1.898)	< 0.0001
IL-8	1.000	(0.999, 1.000)	0.1466
CORT	0.993	(0.986, 1.000)	0.0541

Table 2.8 Odds ratio estimates with associated 95% confidence interval and P-values for exit velocity (m/s) and chute score at first and second processing (EV1, EV2, CS1, CS2), and circulating interleukin 8 (IL-8, pg/mL) and cortisol (CORT, ng/mL) concentrations at first processing with post-harvest USDA quality grade for Years 1 and 2 combined.

	USDA Quality Grade		
Trait	**Odds Ratio Estimate**	**95% Confidence Interval**	**P-value**
EV1	0.976	(0.873, 1.090)	0.6626
EV2	0.964	(0.880, 1.055)	0.4276
CS1	1.042	(0.936, 1.160)	0.4557
CS2	0.984	(0.888, 1.090)	0.7533
IL-8	1.000	(0.999, 1.000)	0.2677
CORT	1.006	(1.000, 1.012)	0.0564

Table 2.9 Odds ratio estimates with associated 95% confidence interval and P-values for exit velocity (m/s) and chute score at first and second processing (EV1, EV2, CS1, CS2), and circulating interleukin 8 (IL-8, pg/mL) and cortisol (CORT, ng/mL) concentrations at first processing with steer mortality in the feedlot for Years 1 and 2 combined.

	Mortality		
Trait	**Odds Ratio Estimate**	**95% Confidence Interval**	**P-value**
EV1	1.107	(0.870, 1.409)	0.4092
EV2	0.978	(0.784, 1.220)	0.8411
CS1	1.176	(0.936, 1.477)	0.1629
CS2	0.901	(0.698, 1.163)	0.4217
IL-8	1.001	(1.000, 1.001)	0.1500
CORT	1.005	(0.990, 1.019)	0.5248

Table 2.10 Correlation matrix with the partial correlation coefficients and associated significance of exit velocity (EV) and chute score (CS) at placement (1) and reimplantation (2).

Trait	CS 1[1]	CS 2[2]	EV 1[3]	EV 2[4]
CS 1	1.000			
CS 2	0.2351***	1.000		
EV 1	0.1406***	0.1803***	1.000	
EV 2	0.1373***	0.2223***	0.4448***	1.000

*P < 0.05; **P < 0.01; and ***P < 0.001.

Table 2.11 Partial correlation coefficients of chute score (CS) and exit velocity (EV) at placement (1) and reimplantation (2) with immune, performance, and carcass traits in beef cattle

Trait	CS 1	CS 2	EV 1	EV 2
Cortisol (ng/mL) at placement	0.072**	0.075**	0.037	0.112***
Interleukin 8 (pg/mL) at placement	-0.011	-0.026	0.016	0.044
Weight at placement (kg)	0.026	0.013	-0.008	-0.026
Weight at second processing (kg)	-0.012	-0.012	-0.045	-0.105***
Weight at third processing (kg)	-0.023	-0.040	-0.059*	-0.111***
Gain from first to second processing (kg)	-0.034	-0.025	-0.048	-0.108***
Gain from second to third processing (kg)	-0.022	-0.049*	-0.034	-0.034
Gain from first to third processing (kg)	-0.045	-0.058*	-0.066*	-0.117***
Hot carcass weight (kg)	0.019	-0.024	-0.037	-0.080***
Yield grade	-0.028	-0.038	-0.015	-0.072**
Marbling score	-0.064**	-0.014	-0.018	-0.045
Ribeye area (cm^2)	0.023	0.009	-0.009	0.013
Average lung score	0.038	0.004	-0.009	-0.011

*P < 0.05; **P < 0.01; and ***P < 0.001.

Table 2.12 Least squares means (± SE) with associated P-values for the change in chute score (CS change) between first and second processing by average chute score at placement (CS)

CS	CS Change	P-value
1	0.90 ± 0.12	< 0.0001
1.5	0.29 ± 0.08	0.0344
2	0.10 ± 0.05	< 0.0001
2.5	-0.16 ± 0.05	< 0.0001
3	-0.49 ± 0.04	< 0.0001
3.5	-0.77 ± 0.06	0.0006
4	-1.02 ± 0.06	0.0015
4.5	-1.14 ± 0.11	< 0.0001
5	-1.50 ± 0.24	< 0.0001

Table 2.13 Contingency table of chute scores at first and second processing for Years 1 and 2 combined.

	CHUTE2										
CHUTE1	**1**	**1.5**	**2**	**2.5**	**3**	**3.5**	**4**	**4.5**	**5**	**6**	**Total**
1	11	4	30	6	9	2	3	0	0	0	65
1.5	12	6	61	16	35	4	3	1	0	0	138
2	40	37	256	47	143	27	29	0	1	0	580
2.5	23	21	188	55	130	25	15	2	2	0	461
3	47	31	297	116	259	58	53	4	5	1	871
3.5	14	10	93	26	89	20	28	4	6	0	290
4	18	14	83	27	88	25	35	5	11	0	306
4.5	3	5	17	7	13	7	14	6	7	0	79
5	2	1	0	2	3	1	6	0	2	0	17
Total	170	129	1025	302	769	169	186	22	34	1	2807

Table 2.14 Least squares means (±SE) for change in chute score (CS) and exit velocity (EV) for steers that were treated and not treated for bovine respiratory disease (BRD) prior to feedlot entry and treated or not treated for BRD after feedlot entry for Years 1 and 2 combined.

	Pre-entry			Post-entry		
Trait	**Treated**	**Not Treated**	**P-value**	**Treated**	**Not Treated**	**P-value**
CS Change	-0.2770	-0.3249	0.4063	-0.3181	-0.2837	0.3369
EV Change	0.0591	0.0081	0.5405	0.0600	0.0638	0.4819

References

Bateman, K. G., W. Martin, P. E. Shewen, and P. I. Menzies. 1990. An evaluation of antimicrobial therapy for undifferentiated bovine respiratory disease. Can. Vet. J. 31:689-696.

BIF. 2002. Beef Improvement Federation Guidelines: Behavioral Traits. Beef Improvement Federation. Accessed Sept. 23, 2011. http://www.beefimprovement.org/library/06guidelines.pdf

Buhman, M.J.; L.J. Perino, M.L. Galyean, T.E. Wittum, T. H. Mntgomery, and R. S. Swingle. 2000. Association between changes in eating and drinking behaviors and respiratory tract disease in newly arrived calves at a feedlot. Amer. J. Vet. Res. 61: 1163-1168.

Burrow, H. M., G. W. Seifert, and N. J. Corbet. 1988. A new technique for measuring temperament in cattle. Proc. Aust. Soc. Anim. Prod. 17:154-157.

Café, L.M., D.L. Robinson, D.M. Ferguson, B.L. McIntyre, G.H. Geesink, and P.L. Greenwood. 2011. Cattle temperament: Persistence of assessments and associations with productivity, efficiency, carcass and meat quality traits. J. Anim. Sci. 89:1452-1465.

Cooke, R. F., J. D. Arthington, D. B. Araujo, and G. C. Lamb. 2009. Effects of acclimation to human interaction on performance, temperament, physiological responses, and pregnancy rates of Brahman-crossbred cows. J. Anim. Sci. 87:4125-4132.

Curley, K. O., Jr., J. C. Paschal, T. H. Welsh, Jr., and R. D. Randel. 2006. Technical note: Exit velocity as a measure of cattle temperament is repeatable and associated with serum concentration of cortisol in Brahman bulls. J. Anim. Sci. 84:3100-3103.

Curley, K. O., Jr., D. A. Neuendorff, A. W. Lewis, J. J. Cleere, T. H. Welsh, Jr. and R. D. Randel. 2007. Functional characteristics of the bovine hypothalamic-pituitary-adrenal axis vary with temperament. Hormones and Behavior 53:20-27.

Fell, L.R., I.G. Golditz, K.H. Walker, and D. L. Watson. 1999. Associations between temperament, performance, and immune function in cattle entering a commercial feedlot. Aust. J. Exp. Agric. 39: 795-802.

Gardner, B. A., H. G. Dolezal, L. K. Bryant, F. N. Owens, and R. A. Smith. 1999. Health of finishing steers: Effects on performance, carcass traits, and meat tenderness. J. Anim. Sci. 77: 3168-3175.

Grandin, T. 1993. Behavioral agitation during handling of cattle is persistent over time. Appl. Anim. Behav. Sci. 36:1-9.

King, D. A., C. E. Schuehle Pfeiffer, R. D. Randel, T. H. Welsh Jr., R. A. Oliphint, B. E. Baird, K.O. Curley Jr., R. C. Vann, D. S. Hale, J. W. Savell. 2006. Influence of animal temperament and stress responsiveness on the carcass quality of beef tenderness of feedlot cattle. J. Meat Sci. 74:546-556.

Martin, S.W., K.G. Bateman, P. E. Shewen, S. Rosendal, J.G. Bohac, M. Thorburn. 1990. A group level analysis of the associations between antibodies to seven putative pathogens and respiratory disease and weight gain in Ontario feedlot calves. Can. J. Vet. Res. 54: 337-342.

McNeill, J. W., J. C. Paschal, M. S. McNeill and W. W. Morgan. 1996. Effects of morbidity on performance and profitability of feedlot steers. J. Anim. Sci. 74(Suppl. 1): 135 (Abstr.).

Oliphint, R. A. 2006. Evaluation of the inter-relationships of temperament, stress responsiveness and immune function in beef calves. M.S. Thesis. Texas A &M University, College Station.

Reinhardt, C. D., W. D. Busby and L. R. Corah. 2009. Relationship of various incoming cattle traits with feedlot performance and carcass traits. J. Anim. Sci. 87: 3030-3042.

Schneider, M. J., R. G. Tait, Jr., W. D. Busby, and J. M. Reecy. 2009. An evaluation of bovine respiratory disease complex in feedlot cattle: Impact on performance and carcass traits using treatment records and lung lesion scores. J. Anim. Sci. 87:1821-1827.

Shuster, D.E., E.K. Lee, M. E. Kehrli Jr. 1996. Bacterial growth, inflammatory cytokine production, and neutrophil recruitment during coliform mastitis in cows within ten days after calving, compared with cows at midlactation. Am. J. Vet. Res. 57: 1569-1575.

Shuster, D. E., M. E. Kehrli Jr., P. Rainard, M. Paape. 1997. Complement fragment C5a and inflammatory cytokines in neutrophil recruitment during intramammary infection with Escherichia coli. Infect. Immun. 65: 3286-3292.

Snowder, G. D., L. D. Van Vleck, L.V. Cundiff, and G. L. Bennett. 2006. Bovine respiratory disease in feedlot cattle: Environmental, genetic, and economic factors. J. Anim. Sci. 84:1999-2008.

Stahringer, R.C., R. D. Randel, and D. A. Neuendorff. 1990. Effects of naloxone and animal temperament on serum luteinizing hormone and cortisol concentrations in seasonally anestrous Brahman heifers. Theriogenology 34:393-406.

Tobler, A., R. Meier, M. Seitz, B. Dewald, M. Baggiolini, and M.F. Fey. 1990. Glucocorticoids downregulate gene expression of GM-CSF, NAP-1/IL-8, and IL-6, but not of M-CSF in human fibroblasts. Blood 79:45-51.

Weaber, R. L. and F. E. Creason. 2010. Phenotypic and genetic relationships among beef cattle temperament and production traits. Proc. of XV Baltic Animal Breeding and Genetics Conference. May 31, 2010. Riga, Latvia.

Chapter 3 - Estimation of genetic parameters for temperament, immune, and carcass traits in beef cattle

Introduction

Cattle temperament has historically influenced selection decisions simply for convenience and safety of handling. However, temperament may have a genetic association with economically relevant traits. An understanding of the genetic basis of temperament traits may influence producers' breeding decisions to result in more productive animals.

Common measures of cattle temperament include pen scores, chute scores (CS), and exit velocities (EV). Previous literature estimates temperament to be moderately heritable, with heritability estimates ranging from 0.15 to 0.44 (Schrode and Hammack, 1971; Stricklin et al., 1980; Fordyce et al., 1988; Burrow and Corbet, 2000; Kadel et al., 2006; Taxis, 2011), indicating the genetic progress in temperament can be made by selection. If genetic correlations are found between temperament and production traits or immunological factors, it may aid cattle breeders in producing profitable cattle through increased performance or carcass merit. Such relationships have been found between EV and HCW (r = -0.54), between EV and marbling score (MS) (r = 0.10), and between EV and yield grade (YG) (r = -0.22) (Nkrumah et al, 2007). Weaber et al. (2006) also found a correlation between weight gain and EV in crossbred steers post-weaning (r = - 0.24; $P < 0.02$).

Phenotypic correlations have been found between temperament and cortisol concentrations, suggesting that more temperamental cattle are more easily stressed (Curley et al., 2006; Cooke et al., 2009). Stress can inhibit immune function by inhibiting the cell-mediated immune response (Jain et al., 1991; Pawlikowski, 1988). Cytokines, such as interleukin-8 (IL-8), are necessary for a cell-mediated immune response to bacterial infections (Goubau et al., 2000). Reinhardt et al. (2009) found that animals that have respiratory disease in the feedlot as shown by lung lesions post-harvest had decreased ADG, HCW, longissimus muscle area, and fat thickness.

Bovine respiratory disease (BRD) has been previously estimated to be lowly heritable (Muggli-Cockett et al., 1990; Snowder et al., 2005, 2006, 2007; Heringstad et al., 2008; Schneider et al., 2008). No previous literature reporting genetic correlations with BRD were found.

Although phenotypic relationships have been found between temperament, immune function, and resulting performance and carcass merit in cattle, no previous literature was found elucidating genetic relationships. The purpose of this study was to estimate genetic parameters for temperament, immunological, and carcass traits, and to determine relationships among them.

Materials and Methods

Animals

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

A single ranch source in western Nebraska provided crossbred steers from 3 locations in November of 2007 (Year 1; n=1,551) and 2008 (Year 2; n=1,319). Steers were shipped from their respective units to a commercial feedlot in southeastern Colorado.

Processing

In both years, cattle were processed within 2 days of arrival to the feedlot (d 0). Initial processing included administration of an oral parasiticide (Synanthic, Boehringer Ingelheim, St. Joseph, MO), a pour-on parasiticide (Promectin, Vedco, St. Joseph, MO), and a growth promotant (Year 1: Revalor-IS; Year 2: Revalor XS; Merck, Summit, NJ). Each animal was assigned both a radio frequency and visual identification tag. Ear notches were taken to determine if the animal was persistently infected with bovine viral diarrhea virus, and those that tested positive were removed from the project. At this time, animal weight was recorded and a blood sample (30 mL) was taken. Because Year 1 cattle were not vaccinated upon placement, the feedlot experienced high BRD rates (45% of cattle were treated). To avoid similar problems in Year 2, cattle were vaccinated with Pyramid 2 + Type II BVD and Presponse SQ (Boehringer Ingelheim, St. Joseph, MO) to target *Pastuerella multocida* and *Mannheimia haemolytica*, both of which are key factors in the development of BRD.

At the time of reimplantation (d 73 – d 100), weight and gain since placement were recorded for each animal. Cattle were processed one more time 59 to 70 d post reimplantation, and records from this processing included weight and total gain since placement.

Temperament

Cattle temperament was measured using two metrics: exit velocity (Burrow et al., 1988) and chute score (Grandin, 1993; BIF, 2002) at placement and reimplantation. Upon entrance to the working chute, cattle were subjectively assigned a CS by 2 observers. Chute score assignments were based on a 6-point scale defined by the Beef Improvement Federation (BIF) as follows: 1=calm, relaxed; 2= fairly calm yet restless, stubborn with little tail flicking; 3=moving nervously, struggling moderately to exit, some tail flicking; 4= agitated, quivering and jumping frantically with continuous tail flicking, urinates and defecates in chute; 5= aggressive, extremely agitated, bellowing and jumping excessively jumping and bellowing; 6= obvious attack behavior that is amplified in confined areas (BIF, 2002). Once necessary data were recorded on each animal, they exited the chute. Upon exit, a timer was triggered by the moving animal that measured the total time it took the steer to move 1.83 m. The distance (1.83 m) was then divided by the time to determine the EV of the animal. For both EV and CS, evaluators were positioned in a specific and consistent place to avoid differential influences on animal response.

Harvest

Cattle were harvested between 68 and 97 d after the third processing at JBS Swift and Company plants in Dumas, TX and Greeley, CO in Year 1 and 2, respectively. Slaughter data collected included HCW, a calculated YG, numeric MS, ribeye area (REA), and lung scores (LUNG). Yield grade was determined using a visual assessment of percent kidney, pelvic, and heart fat, REA, HCW, and an adjusted fat thickness.

Lung scores were subjectively assigned by two trained evaluators. Each evaluator appraised the aggregate lung on a scale of 0 to 3: 0= a healthy lung that may showed healed areas; 1=lung damage in less than 5% of the total lung volume, minimal pleuritis; 2= damage from adhesions greater than one anterior ventral lobe; 3= large amount of missing tissue caused by pleuritis, greater than 15% of the total lung volume damaged by adhesions or active lymph nodes. The 2 scores were then averaged out for a final lung score.

BRD Treatment

Commercial feedlot personnel monitored pens once daily to check for cattle exhibiting signs of illness. Steers exhibiting 2 or more signs of BRD (lethargy, nasal and optical discharge, depression, fever, or cough) were treated on site by personnel of the Colorado State University

South Eastern Colorado Research Center (SECRC). Treatment was determined by protocol set by the commercial feedlot and treated cattle were monitored for another 5 to 7 d post-treatment. Those still exhibiting 2 or more clinical signs of BRD at this time were retreated. Once animals no longer showed BRD signs, they were returned to their respective feedlot pens.

Metabolite and Cytokine Assays

Centrifugation of whole blood samples was followed by plasma extraction. Plasma was stored at -80º C until analysis. Plasma cortisol was measured in duplicate following manufacturer's protocol in a commercially available RIA kit (Coat-A-Count; Diagnostic Products, Los Angeles, CA). A standard curve based on 0, 10, 50, 100, 200 and 500 µg/mL of cortisol was used to determine concentration of unknown samples. A high (200 µg/mL) and low (10 µg/mL) control were used to determine intra- and inter-assay coefficients of variability with a minimal detectable concentration of 2 ng/mL. Concentrations of IL-8 were measured in duplicate using human ELISA kits according to the manufacturer's instructions (R&D Systems, Inc., Minneapolis, MN). Human ELISA has previously shown to cross-react with bovine IL-8 (Shuster et al., 1996, 1997; R&D Systems, Inc., Minneapolis, MN). A standard curve based on 0, 31.2, 62.5, 125, 250, 500, 1000, and 2000 pg/mL detected a minimal concentration of 3.5 pg/mL.

Statistical Analysis

Statistical analysis was performed with ASREML (Ver. 3.0, VSN International, Ltd., Hemel Hempstead, UK) on 2,870 animal records. The pedigree file had records of 7,177 animals with up to 7 generations of pedigree, which included 548 sires and 125 paternal grandsires. Data were analyzed using a multiple trait mixed animal model. Fixed effects included contemporary group (n=11) and pre-weaning BRD treatment, and random effects included animal and permanent environment. Contemporary group included initial ranch unit, date of arrival to the feedlot, processing dates, and feedlot pen. Traits analyzed included EV and CS from both placement and reimplantation, cortisol and IL-8 concentrations from feedlot placement, weights from all processing points, all measures of gain, BRD incidence, HCW, REA, YG, MS, andLUNG. Traits were analyzed in pairs. The model used to estimate direct genetic variance components was (Mrode, 2005):

$$\begin{bmatrix} y_1 \\ y_2 \end{bmatrix} = \begin{bmatrix} X_1 & 0 \\ 0 & X_2 \end{bmatrix} \begin{bmatrix} b_1 \\ b_2 \end{bmatrix} + \begin{bmatrix} Z_1 & 0 \\ 0 & Z_2 \end{bmatrix} \begin{bmatrix} u_1 \\ u_2 \end{bmatrix} + \begin{bmatrix} e_1 \\ e_2 \end{bmatrix}$$

Where,

$\boldsymbol{y}$ = a vector of phenotypic observations

$\boldsymbol{X}$ = an incidence matrix containing fixed effects

$\boldsymbol{b}$ = a vector of fixed effects solutions

$\boldsymbol{Z}$ = an incidence matrix containing random effects

$\boldsymbol{u}$ = a vector of random effect solutions

$\mathbf{e}$ = a vector of residual effects

Multivariate analysis of BRD incidence with all measures of temperament and blood parameters was performed to estimate genetic correlations. For each multivariate analysis that included it, BRD incidence was converted to an underlying continuous scale using a probit function. The probit function is the threshold model commonly used in animal breeding (Gionola and Foulley, 1983; Kadarmideen et al., 2000; 2001). The probit link models the probability that an animal experiences BRD incidence [P(y=1)] and is given by:

$$f_i = \Phi^{-1}[\eta_i]$$

where Φ^{-1} is an inverse normal cumulative density function.

The assumed model variance was:

$$var\begin{bmatrix} u_1 \\ u_2 \\ e_1 \\ e_2 \end{bmatrix} = \begin{bmatrix} g_{11}A & g_{12}A & 0 & 0 \\ g_{21}A & g_{22}A & 0 & 0 \\ 0 & 0 & Ir_{11} & Ir_{12} \\ 0 & 0 & Ir_{12} & Ir_{22} \end{bmatrix}$$

Where,

$\boldsymbol{G} = \{g_{ij}\}$ is the additive genetic (co)variance matrix for animal effects

$\boldsymbol{A}$ = the numerator relationship matrix among animals

$\boldsymbol{I}$ = an identity matrix

$\boldsymbol{R} = \{r_{ij}\}$ is the residual (co)variance matrix

The mixed model equation used in this analysis is as follows:

$$\begin{bmatrix} X'R^{-1}X & X'R^{-1}Z \\ Z'R^{-1}X & Z'R^{-1}Z + G_1 * A^{-1} \end{bmatrix}\begin{bmatrix} \hat{b} \\ \hat{u} \end{bmatrix} = \begin{bmatrix} X'R^{-1}y \\ Z'R^{-1}y \end{bmatrix}$$

Where,

$$y = \begin{bmatrix} y_1 \\ y_2 \end{bmatrix}, \hat{b} = \begin{bmatrix} \hat{b_1} \\ \hat{b_2} \end{bmatrix}, \hat{u} = \begin{matrix} \widehat{u_1} \\ \widehat{u_2} \end{matrix},$$

$$X = \begin{bmatrix} X_1 & 0 \\ 0 & X_2 \end{bmatrix}, Z = \begin{bmatrix} Z_1 & 0 \\ 0 & Z_2 \end{bmatrix}, G_1 = \begin{bmatrix} g_{11} & g_{12} \\ g_{21} & g_{22} \end{bmatrix}^{-1}$$

Here, $\hat{\boldsymbol{b}}$ is a vector of fixed effects solutions, $\hat{\boldsymbol{u}}$ is a vector of random animal effect solutions, $\boldsymbol{y}$, $\boldsymbol{X}$ and $\boldsymbol{Z}$ are the same as previously described, $\boldsymbol{G}$ is the additive genetic variance and covariance matrix, $\boldsymbol{A^{-1}}$ is the inverse of the numerator relationship matrix, and $\boldsymbol{R^{-1}}$ is the inverse of the residual (co)variance matrix.

Results and Discussion

Heritabilities

Table 3.1 shows the heritability estimates of both CS at feedlot placement and CS at reimplantation. Burrow and Corbet (2000) previously estimated the heritability of CS to be 0.30 in zebu-derived beef cattle post-weaning. Kadel et al.(2006) performed a study on Brahman, Belmont Red, and Santa Gertrudis steers and heifers post-weaning and reported a similar CS heritability estimate to the current study ($h^2 = 0.19$), although the animals studied were *Bos indicus* breeds. The largest CS heritability estimate reported was 0.44 by Stricklin et al. (1980).

Heritability of EV at feedlot placement is shown in Table 3.1. Previous research provides several estimates of the heritability of EV. Moderate heritabilities of EV have been reported in cattle of *Bos indicus* descent by Burrow and Corbet (2000; $h^2 = 0.35$); Kadel et al. (2006; $h^2 = 0.21$), and Sant'Anna et al. (2012; $h^2 = 0.26$). Weaber et al. (2007) estimated genetic parameters for temperament traits in Angus x Red Angus crossbred steers at the time of feedlot placement. Although the sampled animals from the Weaber et al. (2007) study are most similar to the current study, the heritability estimate is greater ($h^2 = 0.35$). All discussed heritabilities indicate that genetic progress can be made through selection on temperament traits.

Estimated heritability for circulating cortisol concentrations at the time of feedlot placement was lowly to moderately heritable, and blood concentration of IL-8 was moderately heritable (Table 3.1). No previous literature was found reporting heritabilities of IL-8 or cortisol concentrations.

The ability for cattle to acclimate was measured by the change in CS and the change in EV between the first and second processing. Both metrics indicate that an animal's ability to acclimate is lowly heritable. When the acclimation was measured by CS, the estimated heritability of acclimation was 0.11 ± 0.04. When acclimation was measured by EV, the estimated heritability of acclimation was 0.10 ± 0.04. Previous research has not published heritability estimates of acclimation ability. These heritability estimates indicate that selection on an animal's ability to adapt would result in slow genetic progress.

Heritabilities of carcass traits and disease incidence for this dataset, as well as genetic correlations between disease and carcass traits, were previously reported by McAllister (2010).

Genetic Correlations

Tables 3.1 and 3.2 include all genetic correlation estimates, excluding those that were previously reported (McAllister, 2010). All temperament traits were positively correlated with one another (Table 3.1). The weakest genetic correlation among temperament traits was between CS at the time of feedlot placement and EV at reimplantation (Table 3.1). Exit velocities at placement and reimplantation had a stronger genetic correlation than CS at placement and CS at reimplantation (Table 3.1). This may be an artifact of the subjective nature of CS, as subjective measures have less accuracy and consistency than objective measures. Further investigation of these traits showed that correlation between subsequent measures of EV was 0.41 ± 0.02, and the correlation between subsequent measures of CS was 0.17 ± 0.02, indicating that EV was a more repeatable measure of temperament than CS. Thus, a single measure of EV may be an accurate predictor of future temperament.

Genetic potential for greater serum IL-8 concentrations may indicate that the animal will be more genetically predisposed to bacterial infections or that the animal will have a greater cell-mediated immune response to bacterial infections. Further research should be done to estimate basal concentrations of circulating IL-8, which could determine if the following relationships are causative in nature. Exit velocity at feedlot placement and reimplantation had a negative genetic relationship with the circulating IL-8 concentration at the time of placement (Table 3.1). Chute score at placement and reimplantation was not genetically correlated with serum IL-8 (Table 3.1). Circulating IL-8 concentration at feedlot placement had a positive genetic correlation with total gain, HCW, MS, YG and BRD incidence (Table 3.2). Interleukin-8 concentration was

negatively correlated with LUNG, but showed no significant genetic relationship with circulating cortisol concentrations (Table 2). No previous research has reported genetic correlations involving IL-8 concentrations.

Cortisol had no significant genetic relationship with any temperament measure (Table 3.1), nor with any measure of gain (Table 3.2). Initial cortisol concentration had a negative genetic association with HCW and REA, but it had no significant genetic correlation with MS or YG (Table 3.2). This may indicate that animals that are genetically prone to be more stress susceptible generally have genetics for lighter HCW and smaller REA. Cortisol was genetically related to BRD incidence as measured by both LUNG and treatment in the feedlot, however the direction of the relationship was different between the two (Table 2). Incidence of BRD as termed by treatment in the feedlot may be erroneous, as some cattle may not exhibit signs of sickness to the extreme that others do. Thus, LUNG may be a better indicator of true BRD incidence. Results suggest that cattle that are genetically prone to be susceptible to stress are less likely to exhibit signs of BRD, yet are more likely to contract BRD and develop lung lesions. No previous literature was found reporting genetic correlations between cortisol and temperament.

Although no gain measurement was genetically correlated with CS at the time of feedlot placement, gain between the second and third processing had a significant, positive genetic correlation with CS at reimplantation (Table 3.2). Aside from IL-8, the total amount gained between the first and third processing was not significantly genetically associated with any measure of temperament (Table 3.2). Exit velocity at both the first and second processing had a negative genetic relationship with the amount gained between the first two processing periods (Table 3.2). Weight gained between the second and third processing had a positive genetic correlation with EV and CS at the time of reimplantation. This indicates that cattle that are genetically oriented to be less temperamental at the time of feedlot placement will also have genetics to gain more weight between the time of placement and reimplantation. However, cattle that are genetically oriented to be more temperamental at reimplantation will have the genetics to gain more between reimplantation and the third processing period. Reasons for this are unknown. Correlations between EV and amount of gain have been previously reported; Sant' Anna et al. (2012) estimated the genetic correlation between EV and ADG to be -0.13 ± 0.08, which was similar to the second gain period in the current study.

Chute score measures at both time points had positive genetic relationships with REA (Table 3.2), suggesting that cattle with genetics to be more temperamental will have genetics for a greater REA. Initial CS was positively genetically associated with HCW; however, CS at the time of reimplantation indicated no significant genetic correlation with HCW (Table 3.2). Exit velocity at the time of reimplantation had a negative genetic association with HCW, but initial EV did not have a genetic correlation with HCW that was different from zero (Table 3.2). This might indicate that cattle genetically inclined to be more temperamental at the time of feedlot placement will also have larger HCW, whereas those that have greater genetic potential to be temperamental at the time of reimplantation will have a smaller HCW. Similar genetic relationships have been previously reported; Nkrumah et al. (2007) found moderate negative genetic associations between EV and HCW (r = -0.54). Both CS and EV had negative genetic correlations with MS at the time of reimplantation, but neither temperament measure was related to MS upon arrival to the feedlot (Table 3.2). This suggests that cattle that have the genetic potential to be more temperamental at reimplantation will have genetics for less intramuscular fat deposition. Exit velocity has previously been reported to have a genetic correlation with MS of 0.10 (Nkrumah et al., 2007). Placement EV had a moderate genetic relationship with REA, but this relationship did not continue through reimplantation (Table 3.2). Temperament measures at each time point had a negative genetic correlation with YG (Table 3.2). Nkrumah et al. (2007) estimated the genetic correlation between EV and YG to be -0.22.

The only temperament measure that was significantly genetically correlated with LUNG was EV at the time of feedlot placement (Table 3.2). Reinhardt et al. (2009) previously reported no significant phenotypic relationships between temperament measures and lung scores. The only temperament measures that showed a genetic correlation with BRD treatment in the finishing phase were those observed at the time of reimplantation (Table 3.2). Of the 2 temperament measures that did show a genetic correlation with BRD treatment in the feedlot, CS at the time of reimplantation had the strongest genetic relationship with BRD treatment in the feedlot segment, indicating that cattle that have greater genetic potential to be temperamental at the time of reimplantation are also those that are less genetically predisposed to BRD (Table 3.2). Reasons for this are unknown.

Conclusions

Results from this study indicate that blood parameters (with the exception of IL-8) and temperament measures all have negative genetic relationships with BRD treatment in beef cattle. More temperamental cattle do not seem to be inherently more susceptible to BRD incidence in the feedlot segment, but results indicate that cattle with genetics to be more docile are those that are also more genetically susceptible to BRD. Measures of temperament are genetically correlated with one another, and EV is estimated to be more repeatable than CS. Genetic correlations indicate that cattle with genetic potential to be more temperamental will have genetics for greater REA, reduced MS, and reduced YG. Selection for calmer cattle could result in cattle that are genetically predisposed to contracting and exhibiting signs of BRD and that are genetically prone to reduced carcass yield.

Tables

Table 3.1 Heritabilities (on diagonal ± SE) and genetic correlations (above diagonal ± SE) among cortisol concentrations (CORT, ng/mL), interleukin 8 (IL-8, pg/mL), chute score at placement (CS1) and reimplantation (CS2) and exit velocity at placement (EV1) and reimplantation (EV2) in beef cattle

Trait	CORT	IL-8	CS1	CS2	EV1	EV2
CORT	0.23 (0.06)	-0.01 (0.16)	0.07 (0.17)	0.09 (0.19)	-0.11 (0.19)	0.11 (0.16)
IL-8		0.34 (0.07)	-0.04 (0.15)	-0.08 (0.17)	-0.31 (0.17)	-0.22 (0.15)
CS1			0.23 (0.05)	0.37 (0.17)	0.31 (0.17)	-0.02 (0.17)
CS2				0.19 (0.05)	0.23 (0.19)	0.27 (0.17)
EV1					0.17 (0.05)	0.73 (0.11)
EV2						0.27 (0.06)

Table 3.2 Genetic correlations (± SE) between temperament or immune traits and carcass traits in beef cattle

Trait	GAIN1[7]	GAIN2[8]	TOTAL GAIN[9]	HCW[10]	MS[11]	REA[12]	YG[13]	LUNG[14]	BRD[15]
CORT[1]	0.03	-0.19	-0.14	-0.34	-0.06	-0.19	0.08	0.16	-0.68
	(0.16)	(0.21)	(0.18)	(0.17)	(0.14)	(0.18)	(0.16)	(0.31)	(0.22)
IL-8[2]	0.04	0.19	0.18	0.40	0.35	-0.01	0.37	-0.44	0.35
	(0.14)	(0.20)	(0.15)	(0.15)	(0.11)	(0.16)	(0.14)	(0.32)	(0.20)
CS1[3]	-0.16	0.00	-0.14	0.18	-0.01	0.39	-0.21	-0.23	-0.01
	(0.16)	(0.21)	(0.17)	(0.17)	(0.13)	(0.18)	(0.16)	(0.33)	(0.22)
CS2[4]	-0.04	0.28	0.12	0.05	-0.16	0.28	-0.30	-0.28	-0.60
	(0.18)	(0.22)	(0.19)	(0.20)	(0.15)	(0.20)	(0.17)	(0.35)	(0.22)
EV1[5]	-0.23	0.09	-0.14	-0.12	-0.01	0.43	-0.46	0.36	-0.09
	(0.17)	(0.23)	(0.19)	(0.19)	(0.15)	(0.19)	(0.16)	(0.34)	(0.24)
EV2[6]	-0.30	0.36	-0.08	-0.24	-0.14	0.17	-0.29	0.16	-0.34
	(0.15)	(0.20)	(0.16)	(0.17)	(0.13)	(0.17)	(0.14)	(0.29)	(0.21)

[1]CORT= Circulating serum cortisol concentration (ng/mL) at the time of feedlot placement
[2]IL-8= Circulating interleukin 8 concentration (pg/mL) at first processing
[3]CS1= Average chute score at the time of feedlot placement (first processing)
[4]CS 2= Average chute score at the time of reimplantation (second processing)
[5]EV 1= Exit velocity (m/s) at the time of feedlot placement
[6]EV 2= Exit velocity (m/s) at the time of reimplantation
[7]GAIN1= Total amount of weight gained (kg) between feedlot placement and reimplantation
[8]GAIN2= Total amount of weight gained (kg) between reimplantation and the third processing
[9]TOTALGAIN= Total amount of weight gained (kg) between first and third processing
[10]HCW= Hot carcass weight (kg)
[11]MS= Marbling score
[12]REA= Ribeye area (cm^2)
[13]YG= Calculated yield grade
[14]LUNG= Averaged appraised lung score of the aggregate lung post-harvest
[15]BRD= Treatment for BRD in the feedlot

References

BIF. 2002. Beef Improvement Federation Guidelines: Behavioral Traits. Beef Improvement Federation. Accessed Sept. 23, 2011. http://www.beefimprovement.org/library/06guidelines.pdf

Burrow, H. M. and N. J. Corbet. 2000. Genetic and environmental factors affecting temperament of zebu and zebu-derived beef cattle grazed at pasture in the tropics. Aust. J. Agric. Res. 51: 155-162.

Cooke, R. F., J. D. Arthington, D. B. Araujo, and G. C. Lamb. 2009. Effects of acclimation to human interaction on performance, temperament, physiological responses, and pregnancy rates of Brahman-crossbred cows. J. Anim. Sci. 87:4125-4132.

Curley, K. O., Jr., J. C. Paschal, T. H. Welsh, Jr., and R. D. Randel. 2006. Technical note: Exit velocity as a measure of cattle temperament is repeatable and associated with serum concentration of cortisol in Brahman bulls. J. Anim. Sci. 84:3100-3103.

Curley, K. O., Jr., D. A. Neuendorff, A. W. Lewis, J. J. Cleere, T. H. Welsh, Jr. and R. D. Randel. 2007. Functional characteristics of the bovine hypothalamic-pituitary-adrenal axis vary with temperament. Hormones and Behavior 53:20-27.

Fordyce, G., R. M. Dodt, and J. R. Wythes. 1988. Cattle temperaments in extensive beef herds in northern Queensland. 1. Factors affecting temperament. Aust. J. Exp. Agric. 28:683-687.

Gianola, D., and J. L. Foulley. 1983. Sire evaluation for ordered categorical data with a threshold model. Genet. Sel. Eval. 15:201-224.

Grandin, T. 1993. Behavioral agitation during handling of cattle is persistent over time. Appl. Anim. Behav. Sci. 36:1-9.

Goubau, S., D. W. Morck, and A. Buret. 2000. Tilmicosin does not inhibit interleukin-8 gene expression in the bovine lung experimentally infected with *Mannheimia (Pasteurella) haemolytica*. Can. J. Vet. Res. 64: 238-242.

Jain, R., D. Zwickler, C. Hollander, H. Brand, A. Saperstein, B. Hutchinson, C. Brown and T. Audhya. 1991. CRF modulates the immune response to stress in the rat. Endocrinology. 128: 1329-1336.

Kadarmideen, H. N., R. Thompson,and G. Simm. 2000. Linear and threshold model genetic parameters for disease, fertility and milk production in dairy cattle. Anim. Sci. 71:411-419.

Kadarmideen, H. N., R. Rekaya, and D. Geanola. 2001. Genetic parameters for clinical mastitis in Holstein-Freisians: A Bayesian analysis. Anim. Sci. 73:229-240.

Kadel, M. J, D. J. Johnson, H. M. Burrow, H. Graser, and D. M. Ferguson. 2006.Genetics of flight time and other measures of temperament and their value as selection criteria for improving meat quality traits in tropically adapted breeds of beef cattle. Aust. J. Agric. Res. 57: 1029-1035.

McAllister, C. M. 2010. Genetics of bovine respiratory disease in feedlot cattle. M.S. Thesis. Colorado State Univ., Fort Collins.

Mrode, R. A. 2005. Linear Models for the Prediction of Animal Breeding Values. 2nd ed. CABI Publishing, Cambridge, MA.

Nkrumah, J. D., D. H. Crews, J. A. Basarab, M. A. Price, E. K. Okine, Z. Wang, C. Li, and S. S. Moore . 2007. Genetic and phenotypic relationships of feedling behavior and temperament with performance, feed efficiency, ultrasound, and carcass merit of beef cattle. J. Anim. Sci. 85: 2382-2390.

Pawlikowski , M., P. Zelakowski, K. Dohler, and H. Stepien. 1988. Effects of two neuropeptides somatoliberin and CRF on human lymphocyte natural killer activity. Brain Behav. Immun. 2: 50-56.

Reinhardt, C. D., W. D. Busby and L. R. Corah. 2009. Relationship of various incoming cattle traits with feedlot performance and carcass traits. J. Anim. Sci. 87: 3030-3042.

Sant'Anna, A. C., M. J. R. Paranhos da Costa, F. Baldi, P. M. Rueda, and L. G. Albuquerque. 2012. Genetic associations between flight speed and growth traits in Nellore cattle. J. Anim Sci. 90: 3427-3432.

Schrode, R. R. and S. P. Hammack. 1971. Chute behavior of yearling beef cattle. J. Anim. Sci. 33:193 (Abstr.).

Stricklin, W. R., C. E. Heisler, and L. L. Wilson. 1980. Heritability of temperament in beef cattle. J. Anim. Sci. 51(Suppl. 1): 109(Abstr.).

Taxis, T. M. 2011. Phenotypic and genetic effects of disposition on beef tenderness and quality attributes. M. S. Thesis. Univ. of Missouri, Columbia.

Weaber, R. L. and F.E. Creason. 2006. Relationship of two measures of disposition and gain performance of steers. J. Anim. Sci. 84(Suppl. 1):55. (Abstr.)

Weaber, R. L. and F. E. Creason. 2007. Genetic parameter estimates for two measures of disposition. J. Anim. Sci. 85(Suppl. 1): 190. (Abstr.)