THESIS

EVALUATING THE GENETIC RELATIONSHIP BETWEEN HIGH ELEVATION PULMONARY ARTERIAL PRESSURE AND MODERATE ELEVATION FEEDLOT PERFORMANCE AND CARCASS TRAITS

Submitted by

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ABSTRACT

EVALUATING THE GENETIC RELATIONSHIP BETWEEN HIGH ELEVATION PULMONARY ARTERIAL PRESSURE AND MODERATE ELEVATION FEEDLOT PERFORMANCE AND CARCASS TRAITS

The objective of the study was to evaluate if a genetic relationship exists between post weaning pulmonary arterial pressure (PAP) measured at high elevation and traits associated with moderate elevation feedlot performance and carcass traits. For this study, PAP (collected 1992 – 2018; n = 6,898), feedlot performance (2014-2018; n = 558), and carcass data (2001-2018; n = 1627) were obtained from the Colorado State University Beef Improvement Center Angus herd. At an elevation of 2,115 m, post weaning, PAP measurements were collected; subsequently, a selected group of steers were relocated to a moderate elevation feedlot (1,500 m) where feedlot performance data was collected.

Genetic relationships were evaluated with a series of 5-trait models using REML statistical approaches. Traits in this analysis were assigned contemporary groups, fixed effects and a direct genetic random effect. A maternal additive and permanent environmental effect was included to weaning weight in the analysis. The heritability estimate for PAP was 0.20 ± 0.03. Genetic correlations between PAP and feedlot traits were positive, with estimates of 0.32 ± 0.20 (average dry matter intake) and 0.03 ± 0.17 (average daily gain). The strongest genetic correlation between PAP and carcass performance traits were those of rib eye area (-0.30 ± 0.12) and calculated yield grade (0.29 ± 0.12). Genetic correlations between PAP and marbling score, back fat, or hot carcass weight were 0.00 ± 0.13, -0.07 ± 0.13, and 0.14 ± 0.10, respectfully.

These results suggest a favorable genetic relationship exists between PAP and feedlot and carcass traits. As cattle with lower post-weaning PAP move to lower elevation feedlot, they could potently have heavier muscled carcasses compared to their high PAP contemporaries.

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CHAPTER 1

INTRODUCTION AND OBJECTIVE

High altitude disease is induced by hypoxia related pulmonary arterial hypertension (PH). A limited amount of oxygen available in areas of high elevation leads to a severe resistance for the flow of blood as it enters the arteries located in the lungs (Farber and Loscalzo, 2004). Subsequently, this hypoxia-promoted condition can ultimately lead to right side heart failure (RHS) as the right ventricle attempts adapts the increased blood pressure (Crapo et al., 2003; Farber and Loscalzo, 2004; Han et al., 2008). Pulmonary arterial pressure (PAP) is used as a risk identification tool and may be used to confirm the presence and degree of PH (Holt and Callan, 2007). While producers have used PAP to make selection decisions against high PAP in their herd, some herds still experience cattle death due to high altitude disease. Approximately 5% of death loss can be attributed to pulmonary hypertension in cattle at high altitude regions (Holt and Callan, 2007). With the high interest in carcass performance and, it is crucial to evaluate the genetic relationship between PAP and carcass and feed utilization traits.

Previously in the beef industry, selection decisions have focused on improving output and economically relevant traits that involve animal growth and post-harvest quality. While the study of feed efficiency in beef cattle is not a new topic, it has gained popularity as the variation and increase in feed costs account for up to 70% of the cost of production (Shike, 2013). With the increasing world population and a corresponding decrease in available resources, the focus on economically relevant traits such as feed intake has been shown to aid in the financial conservation of the beef industry.

With the rapid increase in commercially available technologies for individual animal monitoring, scientific development in this area in beef cattle through research is becoming more viable. Average daily gain (ADG) and dry matter intake (DMI) are two of the more common and easy to use phenotype for evaluating feedlot performance for genetic evaluations.

Currently, little information exists that evaluates the potential genetic relationship between PAP, feed intake, and carcass traits. Because of this, little is known about the potential impact of selection decisions on PAP and feeder cattle performance traits. As expected progeny differences (EPD) for PAP are becoming commercially available through the American Angus Association and with other breeds developing similar genetic evaluations, it is essential to understand potential antagonisms between PAP and other production traits receiving selection pressure in individual breeding objectives, particularly at high elevations. By conducting additional research on the topic, we can fill the fundamental gaps in knowledge and establish the genetic relationships that exist between the performance traits. Therefore, the objective of this thesis is to determine the existence of a genetic relationships between pulmonary arterial pressure, feedlot performance, and carcass traits of Angus cattle from the Colorado State University Beef Improvement Center.

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CHAPTER 2

REVIEW OF LITERATURE

SECTION 1: PULMONARY ARTERIAL PRESSURE BACKGROUND, INFLUENCES, AND CLINICAL SIGNS

Collection/background:

High altitude disease or brisket disease is caused by hypoxia induced pulmonary arterial hypertension (PH). Limited oxygen available in areas of high altitude leads to chronic hypoxia, causing vasoconstriction in the pulmonary vasculature (Farber and Loscalzo, 2004). This vasoconstriction leads to severe resistance for the flow of blood as it enters the arteries located in the pulmonary arterioles. Subsequently, a chronic hypoxia promoted condition can ultimately lead to right heart side failure as the right ventricle attempts to adapt through cardiac structural remodeling leading to the increase of blood pressure (Crapo et al., 2003; Farber and Loscalzo, 2004; Han et al., 2008).

High altitude disease has traditionally been considered a regional issue in the mountainous areas, mainly affecting cattle who reside at elevations greater than 1,500 m (Shirley et al., 2008). Currently, the Beef Improvement Federation (BIF) has developed a phenotypic score evaluation for PAP to help identify cattle who are at risk of developing pulmonary hypertension applied to various on the elevations at which they may reside (BIF, 2019).

Obtaining a PAP measurement is a chute-side procedure and consists of placing a catheter into the pulmonary artery of a restrained animal to identify the presence of PH (Figure 2.1). Mean PAP is a pressure measurement of the arterial side of the pulmonary circulation

(Crapo et al., 2003). Mean PAP is commonly recorded using a cardio graphic monitor (Chemla et al., 2004; Holt and Callan, 2007; Parasuraman et al., 2016). Mean PAP is not represented as the averages of the sPAP and dPAP measurements and is traditionally calculated as follows:

$$mean\ PAP = \frac{1}{3}\ pulse\ PAP + \frac{2}{3} diastolic\ PAP$$

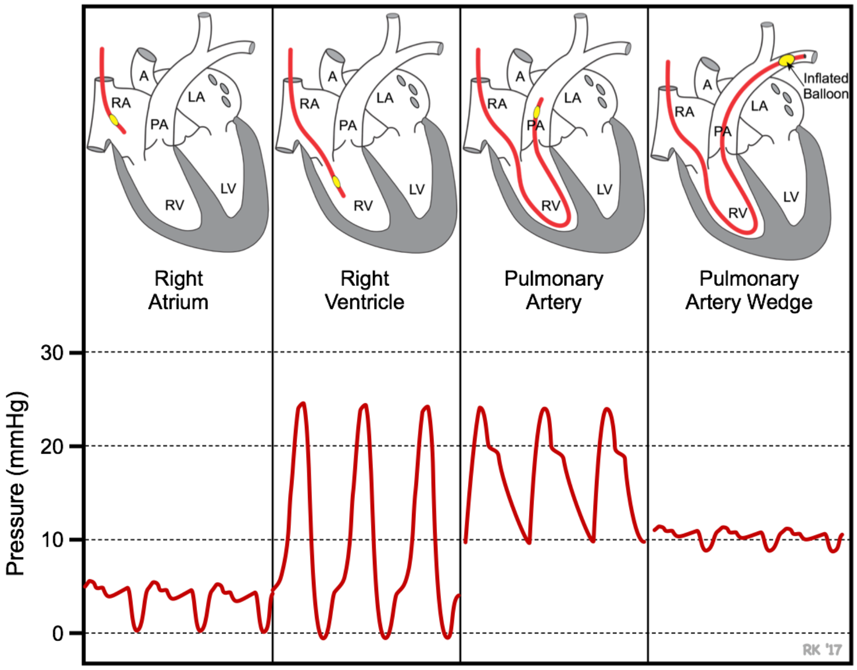

Figure 2.1: A diagram that shows the waveforms as the catheter passes through the right atrium (RA), right ventricle (RV), pulmonary artery, and pulmonary artery wedge. (https://cvphysiology.com/Heart%20Failure/HF008)

Prior to taking a PAP measurement, it is essential that cattle have been subject to a minimum three-week adaptation period at the elevation in which the PAP will be measured. The general procedure for taking a PAP measurement is as follows. First, a flexible catheter is inserted through a 12-gauge 3.5-inch needle that has punctured the proximal jugular vein. The catheter which is connected to a transducer will then be fed through the jugular vein into the right atrium (RA) then through the right ventricle (RV) until it reaches the main pulmonary artery (PA). The mean PAP measurement is collected in the pulmonary artery, and also includes the systolic PAP (sPAP) and diastolic PAP (dPAP) measurements using a cardiographic monitor (Holt and Callan, 2007).

As is presented in Table 2.1, pulmonary arterial pressure measurements below 41 mmHg are considered to be low risk or normal if the animal is over 12 months of age and in an elevation above 1,800m. Conversely, animals whose PAP measurements are greater than 49 mmHg are considered to be high-risk for developing issues related to high PAP (Holt and Callan, 2007).

Table 2.1 Evaluation of pulmonary arterial scores

PAP	Interpretation
30 – 35 mmHg	This score is considered excellent and highly reliable.
36 – 39 mmHg	This score is considered excellent for any animal over the age of 12 months. If the animal is less than 12 months of age, the score is still fairly reliable, but retesting before breeding is suggested.
< 41 mmHg	Scores less than 41 mmHg are reliable measurements in all animals more than 12 months of age. It is recommended that yearling cattle have a PAP measurement less than 41mmHg (depending on altitude of the test). The variation in scores 41 mmHg and above is inconsistent and difficult to predict in some cattle as they age. Any animal measuring 41 mmHg and greater should always be retested before use.
41 – 45 mmHg	This range is acceptable for older animals (i.e., more than 16months of age). Animals less than 16 months scoring in this range should be retested to predict the future PAP of the animal accurately
41 – 45 mmHg	This range is acceptable for older animals (i.e., more than 16 months of age). Animals less than 16 months scoring in this range should be retested to predict the future PAP of the animal accurately
45 – 48 mmHg	This range is acceptable only for older animals that have been in high elevations for an extended period of time. Animals with this score are more susceptible to environmental stresses leading to HMD and should be considered at some risk. Elevation of test site and where the animal lives must be evaluated closely for those in this PAP score range.
> 49 mmHg	Animals that score in this range must always be considered high-risk candidates for developing HMD, not only for themselves but also their offspring. Many animals that have scored in this range have died of HMD. An option for these animals is to move them to a lower elevation for use there. It is also recommended that offspring of these animals never return to high altitude.

These figures are based on cattle tested at or above 1800 m (6000 ft) and12 months of age or greater. If the animal does not meet these criteria then adjustments must be made (Holt and Callan, 2007).

Factors affecting pulmonary arterial pressure:

Elevation:

The ability for an animal to reside in high altitude is crucial to the success of beef operations that are located in elevations over 1,524m. Given the specific nature of PAP, animals at high elevation are at risk for developing elevation related illnesses such as high mountain disease or brisket disease. Susceptibility to high altitude disease is mainly attributed to changes in the cardiovascular physiology of animals that are exposed to higher elevations. Measurements of PAP will tend to increase as animals are moved from lower to higher elevations with every 1,000-foot rise in elevation tends to increase the PAP measurement for cattle 1 to 2 (± 3) mmHg (Holt and Callan, 2007). In a study which evaluated moderate to high elevation effects on PAP in Angus cattle, the raw unadjusted mean PAP measurement was the same for cattle at moderate elevation (< 1,600 m; 43 ± 10.52) and high elevation (>1,600 m; 43 ± 10.87) (Pauling et al., 2018).

As discussed by Neary et al., (2015), the occurrence of pulmonary hypertension in the bovine species is not only an issue for cattle at high altitude. As suggested, it is a combination of the animal's diet, feeding period, age, and altitude that contribute to the presence of pulmonary hypertension. Feedlot heart disease is a condition that resembles high mountain disease and occurs in low to moderate elevation feedlots across the United States (Thomas et al., 2018). Unlike high mountain disease, feedlot heart disease results in right sided heart failure as a result of pulmonary hypertension.

Breed:

Currently, no breed has been shown to be an overall low risk for high PH (Holt and Callan, 2007). Research conducted on 2,041 bulls at the 4-Corners Bull Test reported that the Gelbvieh breed had the lowest PAP measurement, and Simental had the highest PAP when compared to other breeds in the study, detailed results can be seen in table 2.2 (Crawford, 2016). For that study, animals were a small and selected group and likely does not represent breed wide characterization. More research would need to be conducted in order to make a definitive statement regarding breed characteristics regarding PAP measurements.

Table 2.2: Results of model means and coefficients for pulmonary arterial pressure (PAP) by breed in the 4-Corners Bull Test bulls (n = 2,041).

Breed	n[1]	Raw Mean	Estimate	Standard Error
Angus x Gelbvieh	10	44.0	41.3	5.5
Angus	448	47.7	8	4.1
Charolais	282	45.1	8.3	4.3
Composite	296	43.0	4.0	4.3
Gelbvieh	106	37.6	8.3	6.5
Gelbvieh x Herford	7	46.0	9.6	4.4
Herford	518	42.7	3.4	4.2
Limousin	52	43.8	6.9	4.6
Maine Anjou	5	47.2	9.9	7.0
Polled Hereford	114	42.3	5.0	4.2
Red Angus	256	46.7	11.3	4.2
Salers	12	39.5	0.5	5.6
Simmental	83	53.1	13.8	4.5
System 1	216	46.9	10.3	4.3

[1] Number of cattle in each breed group (Natalie Faye Crawford et al., 2016)

Age:

Research conducted by Neary et al. (2013) revealed that PAP measurements will increase as a young calf ages. Since PAP has a tendency to increase with age, animals who are older will typically have a higher PAP when compared to animals at a younger age (Neary et al., 2015). Furthermore, because of the unreliability of PAP score in younger animals, in order to obtain an accurate PAP measurement, the animal must be at least 12 months of age with the preferred age being 16 months or older (Table 2.1) (Holt and Callan, 2007). The age of the animals was shown to be a significant source of variation for PAP with an increase of 0.012 ± 0.007 $\text{mmHg}\cdot\text{d}^{-1}$ (Shirley et al., 2008). An explanation for the positive relationship between age and PAP score can be related to the stiffening of the traditionally more flexible and elastic pulmonary artery (Lammers et al., 2008; Neary et al., 2015)

Environment:

Though there are many environmental factors that influence PAP score, one is the variety of ambient temperatures that accompany the different seasons. A decrease or rapid fluctuation in temperature is more conducive to the development of brisket disease in cattle (Jensen et al., 1976; Shirley et al., 2008). Cold weather induces PH often increasing PAP score from 25% to 55% for cattle who reside in climates where temperatures reach below 0 ºC (Will et al., 1975; Holt and Callan, 2007). Hyperventilation-induced hypoxia caused by a decrease in arterial PO2 with a corresponding increase in arterial PCO2 is the major influential factor contributing to the increased PAP for cattle in cold environments (Holt and Callan, 2007). Jensen et al., (1976) reported in a study across four commercial feedlots that the occurrence of brisket disease was active throughout all seasons but was most common in fall and winter.

Pregnancy:

Cattle could have had higher reported PAP scores while pregnant when compared to similar non-pregnant animals (Moore et al., 1979). Understandably animals who are pregnant are at a higher risk for developing high altitude disease due to pregnancy related PH. Foxworthy et al. (2018) reported that there was no relationship between gestation length and progeny yearling PAP score for heifers who reside in high altitudes.

Genetics:

Current research has shown PAP to be moderately heritable with estimates ranging from 0.26 to 0.34 (Shirley et al., 2008; Crawford et al., 2016; Pauling et al., 2018). Data collected for these estimates are from Angus cattle that dwelled periodically in the states of Wyoming and Colorado. Shirley et al. (2008) expanded the heritably estimates separately for females and males, respectively, 0.38 ± 0.07 and 0.46 ± 0.09. The presented estimates suggested that as producers implement selection pressure on PAP into their breeding objective, there is potential to lower PAP for subsequent generations. Table 2.3 describes previously reported heritability estimates for PAP obtained in cattle.

Table 2.3: Previously reported heritability estimates for pulmonary arterial pressure (PAP) followed by their standard errors

Source	PAP	Breed
Crawford et al., (2016)	0.26 ± 0.03	AN
Shirley et al., (2008)	0.34 ± 0.05	AN
Pauling et al., (2018)	0.34 ± 0.03	AN
Culbertson et al., (2017)	0.37 ± 0.10	AN, CH, SD, GL, SI
Zeng et al., (2014)	0.22 ± 0.04	AN
LeValley, (1978)	0.66 ± 0.21	AN
Enns et al., (1992)	0.46 ± 0.16	AN
Schimmel, (1981)	0.40 ± 0.13	AN
Cockrum et al., (2014)	0.31 ± 0.03	AN

AN = Angus, CH = Charolais, SD = South, Devon, GL = Gelbvieh, SI = Simmental

Additional studies have evaluated the heritability estimates as they vary from animals who are PAP tested at high elevation and those at moderate elevation. In a multi breed study, Culbertson et al., (2017) reported heritability estimates to be moderate for high elevation (>1,800 m) and moderate elevation (<1,800 m) with a strong genetic correlation between traits (0.37 ± 0.10; 0.26 ± 0.08; 0.79 ± 0.23). Additionally, reported estimates for similar studies have been conducted at high elevation (>1,600 m; 0.34 ± 0.03) and moderate elevation (<1,600 m; 0.29 ± 0.09) with a reported genetic correlation of 0.83 ± 0.15 (Pauling et al., 2018). These results suggested that both moderate elevation PAP and high elevation PAP are moderately heritable, with a strong genetic correlation between them. This relationship would suggest that the use of moderate elevation PAP measurements can be an indicator of the animal's PAP measurement as they move to an increased elevation.

Sex:

Shirley et al. (2008) estimated the genetic correlation of PAP scores between males and females to be 0.64 ± 0.12. The increase of PAP per day between male and female was reported at an increase of 0.022 ± 0.008 $\text{mmHg}\cdot\text{d}^{-1}$ of age for females and for males a decrease of 0.004 ± 0.01 $\text{mmHg}\cdot\text{d}^{-1}$ (Shirley et al., 2008). Neary et al. (2013) reported that six-month-old heifers had lower mean PAP measurement than steers of the same age by 4 mmHg. It has been suggested that the variation of PAP between males and females was due to the management differences between sexes rather than physiological reasons (Cockrum et al., 2019).

Clinical Signs:

The effects of pulmonary hypertension can be seen in the beef industry through its relationship with high mountain disease and brisket disease in cattle. As the disease manifests its self within the animal, we see pulmonary vascular remolding as well as cardiac functional and structural changes (Figure 2.2). The narrowing or constriction of the pulmonary arterioles and arterioles in response to the hypoxic condition results in a type three PH. The right ventricle in response to the PH begins to hypertrophy in an attempt to maintain cardiac output and pulmonary profusion of deoxygenated blood into the pulmonary system. After time the right ventricle begins to fail due to overwork and over compensation and enters congestive right ventricular failure, thus leading to poor right ventricle function and ultimately results in death (Tim Holt; personal communication).

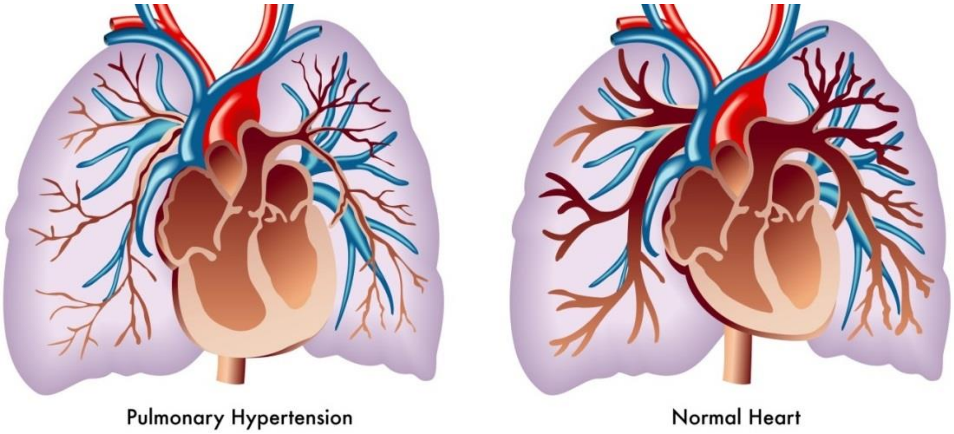

Figure 2.2: visual description of the cardiovascular changes induced by pulmonary hypertension.
https://www.uchicagomedicine.org/forefront/heart-and-vascular-articles/2017/january/uchicago-medicine-earns-pulmonary-hypertension-accreditation

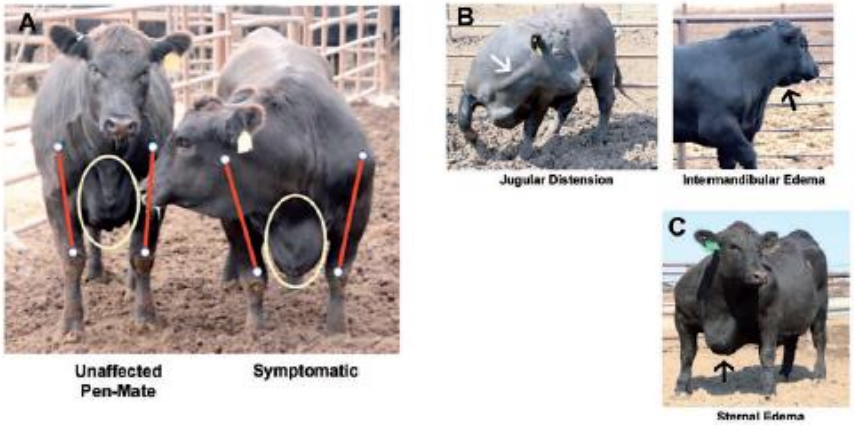

Figure 2.3: Clinical signs of PH and CHF. (a) Symptomatic animal alongside healthy pen-mate. White oval delineates accumulation of fluid in the sternal subcutaneous tissues (symptomatic) compared to no accumulation of fluid in the sternal subcutaneous tissues of the healthy pen-mate. Red lines with closed white circles highlight shoulder abduction in the symptomatic animal compared to the healthy pen-mate due to accumulation of edema fluid in the sternum. (b) Arrow points to striking jugular vein distention (left) and intermandibular edema (right). (c) Arrow points to noteworthy accumulation of fluid in the sternal subcutaneous tissues accompanied by marked shoulder abduction. (Krafsur et al., 2019)

Identifying animals who have PH can be done through visual appraisal in some cases (Figure 2.3). Animals who are suffering from PH are typically lethargic, have decreased appetite, diarrhea, jugular pulsation, and in critical stages, show a large brisket edema. The cause of brisket edema is due to the hydrostatic pressure when the right ventricle goes through failure as well as venous hypertension (Tim Holt; personal communication). Commonly, cases relating to pulmonary hypertension in cattle can be miss diagnosed as a respiratory illness leading to an incorrect mode of treatment.

PULMONARY HYPERTENSION: IMPACT ON THE BEEF INDUSTRY

From an economic standpoint, the effects of pulmonary hypertension directly impact the beef industry. Holt and Callan (2007) reported death losses up to 5% for cattle residing in high elevation, where this translates to the loss of 75,000 cattle and a $60 million loss annually (Williams et al., 2012). Additionally, estimates of 25 to 40% of cattle dying from pulmonary hypertension were reported when cattle were relocated from a lower to a high elevation mountain range (McCormick, 2011).

The expected progeny difference (EPD) for PAP was initially developed by using data from the Tybar Ranch in Carbondale, CO (1,880 m) and the Colorado State University Beef Improvement Center located in Saratoga WY (2,170 m) in 1992 and has been utilized as a tool for selection decisions to some degree since that time (Enns et al., 2011). As visualized in Figure 2.4, utilization of the PAP EPD and proper selection decisions have led to a favorable steady decrease in average PAP EPD values for each respective herd using within herd EPD.

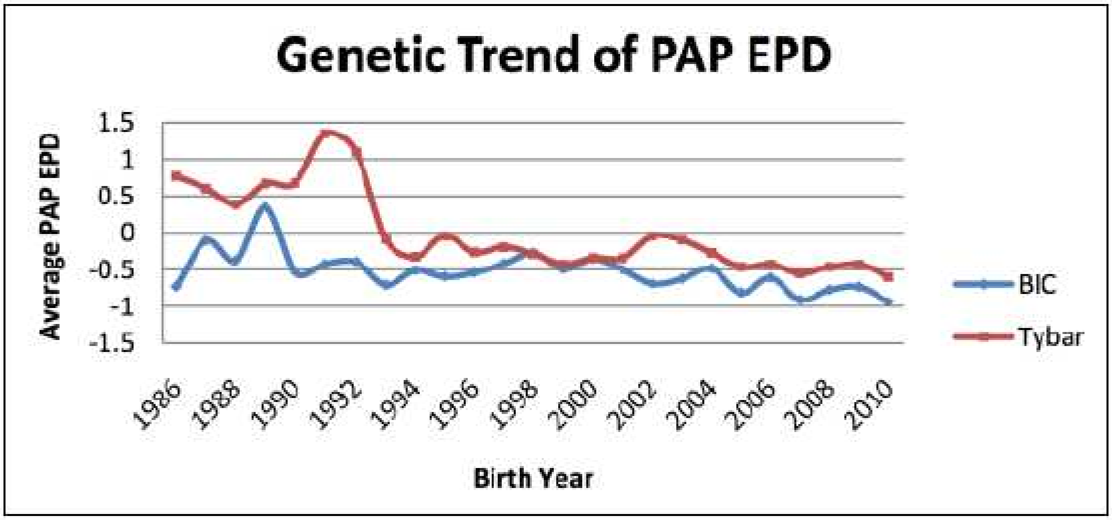

Figure 2.4: Genetic trend in pulmonary artery pressure at the Tybar Ranch (Tybar) and the Colorado State University Beef Improvement Center (CSU-BIC) since selection with EPD began in 1992 (Tybar) and 2002 (CSU-BIC) (Enns et al., 2011).

PULMONARY ARTERIAL PRESSURE AND ITS RELATIONSHIP WITH GROWTH AND FEED UTILIZATION

A genetic correlation is the relationship between the breeding values of a particular trait and the breeding values of another trait (Bourdon, 2000). Numerous relationships between PAP and industry related traits have been analyzed to understand better the potential antagonism that could exist. Genetic correlations between PAP and performance traits related to Angus cattle growth and carcass quality are provided below in Table 2.4.

Schimmel, (1981) and Pauling et al., (2018) reported a negative genetic correlation between PAP with birth weight direct (-0.43 ± 0.29; -0.07 ± 0.13). These results differ from the reported positive genetic correlation between birth weight direct and PAP in other studies (0.40 to 0.22; Crawford et al., 2016; Shirley et al., 2008; Zeng et al., 2014). Shirley et al., (2008) and

Crawford et al., (2016) both reported lowly positive genetic correlations for PAP and birth weight maternal (0.01. ± 0.17, 0.14 ± 0.10). Whereas Pauling et al., (2018) reported a strong positive correlation of 0.54 ± 0.14. The relationship between PAP and weaning weight direct has been reported as a positive correlation by all authors. Yearling weight direct was reported as a strong negative correlation by Schimmel, (1981; -0.75 ± 0.65) however; the remaining authors reported low positive correlations (0.02 to 0.12; Crawford et al., 2016; Pauling et al., 2018; Zeng et al., 2014). The differences in reported estimates could be due to the difference in the populations and methodology (N. F. Crawford et al., 2016; Pauling et al., 2018).

Table 2.4: Previously reported genetic correlations estimates of various traits with pulmonary arterial pressure (PAP) followed by standard error (SE)

Trait	Schimmel, (1981)	Shirley et al. (2008)[1]	Zeng (2013)[2]	Crawford et al., (2016)[2]	Pauling (2017)[2]
BWD	-0.43 ± 0.29	0.49 ± 0.12	0.22	0.15 ± 0.09	-0.07 ± 0.13
BWM	-	0.01 ± 0.17	-	0.14 ± 0.10	0.54 ± 0.14
WWD	0.19 ± 0.34	0.51 ± 0.18	0.16	0.22 ± 0.08	0.14 ± 0.15
WWM	-	-0.05 ± 0.14	0.10	-0.03 ± 0.08	-0.15 ± 0.14
YWD	-0.75 ± 0.65	-	0.11	0.12 ± 0.12	0.02 ± 0.13
YWM	-	-	-	0.00 ± 0.09	-0.06 ± 0.17
PWG	-	-	0.04 ± 0.12	-0.10 ± 0.10	-0.06 ± 0.12
BF	-	-	-	-	-0.03 ± 0.12
REA	-	-	-	-	0.24 ± 0.12
IMF	-	-	-	-	-0.04 ± 0.10
RUMP	-	-	-	-	0.10 ± 0.11

BWD = Birth weight direct, BWM = Birth weight maternal, WWD = Weaning weight direct, WWM = Weaning weight maternal, YWD = Yearling weight direct, YWM = Yearling weight maternal, PWG = Post-weaning gain, BF = Ultrasound back fat, REA = Ultrasound Rib eye area, IMF = Ultrasound intramuscular fat, RUMP = Ultrasound rump fat

[1] Weaning pulmonary arterial pressure

[2] Yearling pulmonary arterial pressure

Currently, limited information exists evaluating the genetic relationship between carcass traits and PAP. Based on results from Pauling et al., (2018), PAP and ultrasound carcass traits tend to be lowly to moderately correlated. This would suggest that selection for lower PAP animals will not have a detrimental negative influence on carcass traits all else equal.

Pulmonary arterial pressure and growth traits

Understanding the potential antagonisms between PAP and production traits such as weight-related traits is crucial due to increasing emphasis in the industry on downward selection for PAP. Previously it has been hypothesized that cattle growth rate and size could potentially be a contributing factor for increased PAP observations (Jensen et al., 1976; Neary et al., 2015). Reported genetic correlations between PAP and weaning weight are low to moderate with the majority of reported values for yearling weight are reported as lowly correlated. These are further described in Table 2.4.

Pulmonary arterial pressure in feedlot cattle

Feedlots in North America have reported the presence of right-side heart failure (RHF) in altitudes below 2,130m. Cattle that enter a feedlot system typically show an increase in mPAP throughout the finishing phase due to the increase in adipose tissue and ruminal engorgement (Neary 2015). The rapid weight gain and high intake of concentrate diets for cattle in feedlots require increased work of the cardiac ventricles and sustained accelerated circulation (Jensen et al., 1976). In a study conducted across four commercial feedlots located at an elevation of 1,600 m, 5.6% of cattle suffered from symptoms associated with PH (Jensen et al., 1976).

Pulmonary arterial pressure and feed utilization traits

Currently, little research exists analyzing the genetic relationship between feedlot efficiency traits and PAP. Maddock et al., (2010) reported in a multibreed study of growing beef cattle that animals with a lower PAP exhibited lower feed intake corresponding to a reduction in feed efficiency when compared to similar animals with a high PAP. Boldt et al., (2014) reported phenotypic correlations between PAP and average daily intake (-0.072) and average daily gain (0.061) suggesting that animals selected for ideal mPAP scores will not experience a decrease in feedlot performance in subsequent generations.

Pulmonary arterial pressure and carcass traits

Relationships between PAP and carcass traits have been evaluated, and a positive trend between increased PAP, growth, and muscle development have been reported (Pauling 2017). Results from Jensen et al., (1976) and Neary et al., (2015) suggest that as fat deposition and muscle mass increase, PAP may increase as well. This could potentially be caused by the increased level of vascular adipose tissue and rapid growth in body size generating stress on the cardiac function and, therefore, could be triggering hypertensive tendencies. This theory is supported by the results of Pauling (2017), who reported a moderate positive genetic correlation between PAP and ultrasound ribeye area (REA; 0.24±0.12).

SECTION 2: FEED UTILIZATION TRAITS

BACKGROUND, COLLECTION, AND INFLUENCES

Background

Beef operations measure how profitable and sustainable a business is by how efficient production is through their cattle's performance. In order for producers to remain competitive, it is crucial to optimize the efficiency of their cattle (Anele et al., 2014). From the years 1977 to 2007, the United States beef industry increased cattle efficiency with a 20% decrease of the number of days required from birth to harvest, a 64% increase in growth rate (kg/hd/day), and a 30% increase in harvest weight (Capper, 2011; Derner et al., 2017). We can attribute this increase of performance to the improvement of management strategies, genetics, and nutrition (Derner et al., 2017). As the popularity of including economically relevant traits such as traits relating to feed utilization or feed efficiency into individual breeding objectives has resulted in a significant amount of attention due to the rise and continuous fluctuation of feed costs. Shike, (2013) reported that feed costs represent 50 to 70% of the total expenses for beef cattle production.

Feed efficiency is used to describe a ratio of inputs to outputs for the livestock industry (Carstens and Tedeschi, 2006). In a study conducted in 1993, ten years of data collected from Kansas feed yards which included daily gain, feed conversion, and the price of corn explained 93 to 94% of the variation for the cost of gain in finishing steers. In this same 1993 study, corn prices accounted for 60% of the variability of profit (Albright et al., 1993). Feed efficiency and utilization traits are controlled by both genetic and nongenetic factors (Hill, 2012). Therefore, the implementation of proper nutrition, management, and genetic selection can improve feed efficiency in beef cattle, and consequently feed costs can be improved (Rolfe et al., 2011). In the past, the beef industry has focused on improving traits associated with outputs such as weight or growth rate. Feed utilization traits can be designed to focus on reducing the level of inputs without compromising outputs (Crews, 2005). Currently, research is needed to identify the genetic variation in traits associated with feed utilization. This research will advance selection to mitigate energy requirements for maintenance in beef cattle (Rolfe et al., 2011).

Individual Animal Intake Collection

Collecting individual animal intake measurements in beef cattle is costly and challenging to measure. Due to the challenges of the collection process, most data that is used and collected is from feeder cattle in settings that resemble a feedlot. Improving feed efficiency for the feedlot industry by 10% would reduce feed costs by $1.2 billion (Shike, 2013). Taking individual animal measurements requires specialized facilities such as the GrowSafe or Calan Gate systems (Figure 2.5).

Figure 2.5: Visualization of monitorization for induvial cattle feed intake A) Calan Gate system with cattle wearing electronic keys around their neck (https://www.ncagr.gov/research/NCDACS-UPRS-ResearchProgram.htm) B) Growsafe feed bunks (https://www.thefencepost.com/news/university-of-wyoming-scientists-explore-benefits-arising-from-weaning-cornstalks-and-cattle-feeding-options/)

Cattle used for measuring individual feed intake and utilization should be weaned, not be younger than 240 days, and at the other end age during the test should not exceed 390 days (Hill, 2012; BIF, 2018). Prior to the start of the feed test, an animal should have a minimum 21-day warm-up period. This warm-up period allows for acclimation to testing facilities as well as the test diet (Hill, 2012; BIF, 2018). Currently, a 70-day minimum test period following the adaptation period is recommended to accurately compute individual feed measurement with body weights collected at 2 or 4-week intervals (Hill, 2012; BIF, 2018). In a study conducted by Culbertson et al., (2015), the testing period reportedly could be shortened to 42 days and still obtain an accurate average daily dry matter intake where to obtain true residual feed intake values, the test would need to be a minimum, of 56 days.

Influences

Many physiological factors influence feed intake in beef cattle, including body composition, age, and size. Body composition has been shown to be an influential factor affecting feed intake as an animal matures. Torres-Vázquez et al., (2018) reported that the genetic correlation between body weights and feed intake from 200d, 400d, and 600d were 0.68 ± 0.09, 0.42 ± 0.11, and 0.61 ± 0.07, respectfully. As a result, the percentage of body fat is used in prediction equations of feed intake for beef cattle (National Research Council, 2000). Chestnut and Brent (1977) reported that animals tend to be more efficient earlier in the feeding period with a steady decrease in feed efficiency as they reach harvest weight (Figure 2.6). For cattle with body fat within the range of 21.3 to 31.5%, it has been suggested that for every 1% increase in body fat there is a decrease in dry matter intake (DMI) by 2.7% (Fox et al., 1988; National Research Council, 2000). Monitoring the relationship between body fat and feed intake can be used as a management tool for identifying animals who have reached ideal harvest conditions (National Research Council, 2000). Age of animals when entering a feedlot can affect feed intake performance. Physically, an older animal can typically consume more feed when compared to younger animals. When comparing yearling cattle versus calves on feed as they enter the feedlot, there is a 10% increase in predicted DMI for yearling cattle (National Research Council, 2000).

Figure 2.6: Visual description of the decrease in feedlot efficiency as cattle reach finishing weight. (https://www.beefresearch.ca/research-topic.cfm/optimizing-feedlot-feed-efficiency-8)

Macek et al., (2011) reported calves that were sourced directly from cow calf operations whose calves were weaned for either 15 or 45 days prior to shipping resulted in an increased average daily gain (ADG) and fewer days on feed during finishing when compared to calves with at weaning. In the same 2011 study, calves with more weaning days prior to shipment were associated with heavier hot carcass weights, but no effect was noticed on finish traits such as marbling score, yield grade, and 12th rib fat thickness (Macek et al., 2011). A Kansas feedlot study conducted in 2003 focused on quantifying the impact of animal health in relation to animal performance. It was concluded that both percentages of animals treated, and death loss impacted average daily gain, feed conversion, as well as the cost of gain (Irsik and Langemeier, 2003).

Environmental influences, such as temperature, have shown to be influential when analyzing the performance of cattle feed efficiency (Hill and Wall, 2017). Reported studies have shown that feed intake decreases as the ambient temperature increases above the thermoneutral zone (National Research Council, 2000). Hill and Wall, (2017) reported in dairy cattle that as temperature and humidity increased, feed intake decreased; however, the efficiency of converting dry matter to milk production increased.

FEED INTAKE MEASUREMENTS

Dry matter intake

Dry matter intake is commonly used in the beef industry and measures the consumption of ingested feedstuff dry matter. For beef cattle operations, DMI could be considered the single most crucial factor that influences feeder cattle performance (Anele et al., 2014). Positive influences of DMI include its ability to be easily translated and utilized by producers (Berry and Pryce, 2013). While DMI is a measure of an animal's feed intake, it is not a direct measurement of efficiency. However, DMI aids in the calculation of feed efficiency measurements that are commonly used in the livestock industry, such as feed conversion ratio (Berry and Pryce, 2013). Additionally, DMI can be used in diet formulation and calculation of nutrient requirements (National Research Council, 2000; Anele et al., 2014). The majority reported heritability estimates for DMI (Table 2.5) fall into the range of moderate to high, indicating potential for genetic improvement.

Table 2.5: Literature reported heritability estimates followed by the standard error for Dry Matter Intake (DMI)

Source	DMI	Breed
Arthur et al., (1997)	0.59 ± 0.07	AN, HE, Polled HE, SH
Arthur et al., (2001)	0.39 ± 0.03	AN
Arthur et al., (2001)	0.48 ± 0.04	CH
Bishop, (1992)	0.30 ± 0.09	HE
Brown and Gacula, (1964)	0.43 ± 0.18	AN, HE, SH
Crowley et al., (2010)	0.49 ± 0.06	AN, CH, HE, LI, SI
Diaz et al., (2014)	0.39 ± 0.03	Multibreed
Elzo et al., (2009)	0.42 ± 0.13	AN, BR, BRA, Crossbreed
Fan et al., (1995)	0.24 ± 0.11	AN, HE
Herd and Bishop, (2000)	0.31 ± 0.08	HE
Hoque et al., (2009)	0.36 ± 0.09	WA
Lancaster et al., (2009)	0.48 ± 0.14	BRA
Mavrogenis et al., (1978)	0.44 ± 0.25	HE
Mrode et al., (1990)	0.06 ± 0.05	HE
Nkrumah et al., (2007)	0.54 ± 0.15	COM
Robinson and Oddy, (2004)	0.27 ± 0.06	Tropically adapted
Rolfe et al., (2011)	0.40 ± 0.02	Crossbreeds
Sasaki et al., (1982)	0.32 ± 0.08	WA
Schenkel et al., (2004)	0.44 ± 0.06	AN, BRA, CH, HE, LI, SI
Zhang et al., (2017)	0.20 ± 0.06	AN, Crossbreeds

AN = Angus, BR = Brahman, BRA = Brangus, CH = Charolais, COM = Composite, HE = Hereford, LI = Limousin, RAN = Red Angus, SH = Shorthorn, SI = Simmental, WA = Wagyu

Average daily gain

Average daily gain is considered to be an essential trait that reflects cattle efficiency as well as economic benefits (Xu et al., 2019). White and Capper, (2013) reported that improving feed efficiency through the trait ADG would lead to improved economic viability, environmental impact, and social acceptability of the beef industry. This is because when the environmental impact decreases, there is an increase in the social acceptability of beef products which shifts the supply curve, maintaining economic viability (White and Capper, 2013). Average daily gain is the measure of the animal's weight gain per day and is calculated by the regression of weight on the number of days on feed (P. F Arthur et al., 2001). Further described in Table 2.6 are reported heritability estimates for ADG most of which are in the range of moderate to high.

Table 2.6: Literature reported heritability estimates followed by the standard error for Average Daily Gain (ADG)

Source	ADG	Breed
Arthur et al., (1997)	0.41 ± 0.08	AN, HE, Polled HE, SH
Arthur et al., (2001)	0.34 ± 0.04	CH
Arthur et al., (2001)	0.28 ± 0.04	AN
Crowley et al., (2010)	0.30 ± 0.06	AN, CH, HE, LI, SI
Diaz et al., (2014)	0.32 ± 0.04	Multibreed
Fan et al., (1995)	0.26 ± 0.20	AN, HE
Herd and Bishop, (2000)	0.38 ± 0.10	HE
Lancaster et al., (2009)	0.21 ± 0.12	BRA
MacNeil et al., (1991)	0.38 ± 0.16	AN, HE
Nkrumah et al., (2007)	0.59 ± 0.17	COM
Robinson and Oddy, (2004)	0.23 ± 0.06	Tropically adapted
Rolfe et al., (2011)	0.26 ± 0.10	Crossbreeds
Schenkel et al., (2004)	0.35 ± 0.03	AN, BRA, CH, HE, LI, SI
Zhang et al., (2017)	0.09 ± 0.04	AN, Crossbreeds
Kelly et al., (2019)	0.29 ± 0.04	Multibreed

AN = Angus, BRA = Brangus, CH = Charolais, COM = Composite, HE = Hereford, LI = Limousin, SH = Shorthorn, SI = Simmental

RELATIONSHIP BETWEEN FEED UTILIZATION TRAITS AND OTHER PERFORMANCE TRAITS

While looking to improve cattle gain and intake, understanding the potential antagonistic relationship with other production traits is crucial because of the increase in popularity with breed associations for genetic evaluations (Schenkel et al., 2004). Ideally, breeders would aim to select for more efficient animals without affecting other significant production and survival traits. The genetic correlations between ADG and DMI are reported within the range of moderate to high and are provided below in Table 2.7.

Table 2.7: Literature reported genetic correlations followed by their standard error (SE) between average daily gain (ADG) and dry matter intake (DMI)

Source	Estimate	Breed
Arthur et al., (2001)	0.54 ± 0.06	AN
Robinson and Oddy, (2004)	0.87 ± 0.05	Tropically adapted
Arthur et al., (2001)	0.39 ± 0.08	CH
Rolfe et al., (2011)	0.56 ± 0.16	Crossbreed
Nkrumah et al., (2007)	0.87 ± 0.09	COM
Berry and Crowley, (2013)	0.78 ± 0.02	Multibreed
Diaz et al., (2014)	0.32 ± 0.11	Multibreed

AN = Angus, CH = Charolais, COM = Composite

Feedlot and carcass performance

Hands et al., (2012) reported a strong inter-relationship between ADG and other feedlot performance traits such as yield grade and quality grade. These results suggested that producers can select for and raise high performing cattle without sacrificing carcass quality. The reported genetic correlations between feed utilization and carcass traits are provided below in Table 2.8.

In a 2013 study involving both Angus and Charolais cattle, it was concluded that the energy requirement for growing steers to deposit fat might be an essential factor for determining feed efficiency. This hypothesis was supported when early-maturing steers showed an increase in daily DMI and a decrease in ADG when compared to late-maturing steers in the same test (Mao et al., 2013). Many studies have looked at the variation of feedlot performance traits as they are associated with weaning age. Weaning age has previously been hypothesized to affect feedlot performance with early weaned calves being more efficient within the feedlot setting (Peterson et al., 1987; Myers et al., 1999; Arthington et al., 2005). Feedlot performance of steers who were weaned early had an increased number of days on feed but showed improved gain and a an overall reduction in days to harvest (Myers et al., 1999). Alternatively, Wolcott et al., (2010) reported that there was no effect of weaning age as it related to feedlot performance for feed utilization traits.

Table 2.8: Carcass traits and feed intake genetic correlations followed by their standard errors

Trait	ADG	DMI	Breed	Source
RIB	-	0.27 ± 0.05[1]	AN	Arthur et al., (2001)
RIB	-	0.61 ± 0.11	Tropically Adapted	Robinson and Oddy, (2004)
RIB	0.23 ± 0.17	0.11 ± 0.15	AN	Torres-Vázquez et al., (2018)
P8	-	0.14 ± 0.05[1]	AN	Arthur et al., (2001)
P8	-	0.59 ± 0.10	Tropically Adapted	Robinson and Oddy, (2004)
P8	0.27 ± 0.14	0.20 ± 0.12	AN	Torres-Vázquez et al., (2018)
CEMA	-	0.43 ± 0.07[1]	AN	Arthur et al., (2001)
CEMA	-	0.23 ± 0.16	Tropically Adapted	Robinson and Oddy, (2004)
CEMA	0.06 ± 0.15	−0.05 ± 0.13	AN	Torres-Vázquez et al., (2018)
IMF	-	0.39 ± 0.14	Tropically Adapted	Robinson and Oddy, (2004)
IMF	0.15 ± 0.19	-	Multibreed	Kelly et al., (2019)
IMF	0.11 ± 0.15	0.30 ± 0.12	AN	Torres-Vázquez et al., (2018)
CWT	0.71 ± 0.09	0.73 ± 0.07	AN	Torres-Vázquez et al., (2018)
CWT	-	0.16 ± 0.17	AN, CH, HE, LI, SI	Crowley et al., (2011a)
CWT	0.22 ± 0.13	-	AN, CH, HE, LI, SI	Crowley et al., (2011b)
CWT	0.30 ± 0.11	-	Multibreed	Kelly et al., (2019)

[1] Ultrasound measurement

ADG = Average daily gain, DMI = Dry matter intake, RIB = subcutaneous fat depths at the 12th/13th rib, P8 = Rump P8 fat depth, CEMA = Carcass eye muscle area, IMF = Intramuscular fat, CWT = Carcass weight, AN = Angus, CH = Charolais, HE = Herford, LI = Limousin, SI = Simmental

SECTION 3: GENETIC EVALUATION OF PAP AND FEED UTILIZATION

Multivariate models

While the univariate model utilizes information from one trait, the multivariate model uses information from multiple traits and typically a genetic and residual relationship amongst the traits. Traditionally, selection for livestock involves evaluating at many different traits that hold performance or economic importance. Additionally, many of these are genetically and/or phenotypically related. Multiple trait evaluation is considered ideal because of its ability to account for the genetic relationship between traits (Mrode, 2014). Another advantage of using the multi-trait model is the decrease in prediction error variance (PEV) and a corresponding increase in accuracy (Schaeffer, 1984). The more drastic decrease in PEV, the greater the absolute difference between the residual and the genetic correlations (Schaeffer, 1984; Thompson and Meyer, 1986; Mrode, 2014). Traits that benefit the most from the use of a multivariate analysis are those that are more lowly heritable. This increase in accuracy for traits with lower heritabilities is due to the increase in information that stems from the residual covariance between traits (Thompson and Meyer, 1986; Mrode, 2014).

In addition, operations can avoid introducing culling bias in their selection decisions through use of a multivariate analysis (Mrode, 2014). Due to the nature of the multivariate analysis, prediction of breeding values for non-recorded or minimally observed traits that are correlated with each other is possible (Schaeffer, 1984). Culling bias can be removed because it includes the last completely reported trait as a genetically related trait (Mrode, 2014). For example, when making a selection decision for yearling weight, it would be essential to incorporate weaning weight to the model in order to accurately select animals and reduce a bias due to sequential culling (Mrode, 2014).

Below is the multivariate model shown in matrix notation (Mrode, 2014).

$$\begin{bmatrix} \boldsymbol{y_1} \\ \boldsymbol{y_2} \end{bmatrix} = \begin{bmatrix} \boldsymbol{X_1} & \boldsymbol{0} \\ \boldsymbol{0} & \boldsymbol{X_2} \end{bmatrix} \begin{bmatrix} \boldsymbol{\beta_1} \\ \boldsymbol{\beta_2} \end{bmatrix} + \begin{bmatrix} \boldsymbol{Z_1} & \boldsymbol{0} \\ \boldsymbol{0} & \boldsymbol{Z_2} \end{bmatrix} \begin{bmatrix} \boldsymbol{u_1} \\ \boldsymbol{u_2} \end{bmatrix} + \begin{bmatrix} \boldsymbol{e_1} \\ \boldsymbol{e_2} \end{bmatrix},$$

Where $\mathbf{y}_\mathbf{i}$ was a vector of observations for the traits corresponding to the *ith* trait, $\mathbf{X}_\mathbf{i}$ and $\mathbf{Z}_\mathbf{i}$ were known incidence matrices relating observations in $\mathbf{y}$ to levels of fixed effects in $\boldsymbol{\beta}$, and random solutions in $\mathbf{u}$, respectively, and $\mathbf{e}_\mathbf{i}$ was a vector of random residuals. Variances and means for the random effects that were included in the model are as follows:

$$\boldsymbol{Var\begin{bmatrix} u_1 \\ u_2 \end{bmatrix} = \begin{bmatrix} \sigma_1^2 & \sigma_{12} \\ \sigma_{21} & \sigma_2^2 \end{bmatrix} \otimes A \; and \; Var\begin{bmatrix} e_1 \\ e_2 \end{bmatrix} = \begin{bmatrix} \sigma_{e_1}^2 & \sigma_{e_{12}} \\ \sigma_{e_{21}} & \sigma_{e_2}^2 \end{bmatrix} \otimes I \; and \; E\begin{vmatrix} \mu \\ e \end{vmatrix} = \begin{vmatrix} 0 \\ 0 \end{vmatrix}}$$

Where **A** was Wright’s numerator relationship matrix, and **I** was an identity matrix of whose order was equal to the number of animals with each respective phenotype (Wright, 1922).

There are both advantages and disadvantages to the use of the multivariate analysis. Multivariate analysis takes longer computationally to solve and utilizes more memory and disk storage when compared to univariate analysis. Additionally, the key to a successful multivariate analysis is in the accurate estimation of both the genetic and residual correlations which can be problematic with smaller data sets (Mrode, 2014).

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CHAPTER 3

EVALUATING THE RELATIONSHIP BETWEEN PULMONARY ARTERIAL PRESSURE, FEEDLOT PERFORMANCE, AND CARCASS TRAITS

INTRODUCTION

In geographical regions above 1,500 m, high altitude disease (HAD) or brisket disease is a condition in which approximately 5% of cattle die annually (Holt and Callan, 2007; Shirley et al., 2008). For cattle operations at high elevation, a limited amount of available oxygen can lead to acute hypoxia, causing vasoconstriction in the pulmonary vasculature (Farber and Loscalzo, 2004). Pulmonary arterial pressure (PAP) is used as an indicator trait to identify cattle who could potentially succumb to brisket disease and are thereby experiencing pulmonary hypertension (Holt and Callan, 2007). High PAP scores in feedlot cattle have previously been associated with stress on the cardiac ventricles and sustained increased circulation caused by the high intake of concentrate diets and rapid weight gain (Jensen et al., 1976). Additionally, cattle entering the feedlot could be at higher risk of developing PH due to an increase in adipose tissue and ruminal engorgement (Neary et al., 2015).

Within the cattle industry, feedstuffs are considered relevant to the beef industry as feed costs represent up to 70% of the total cost of production (Shike, 2013). Through genetic improvement, improving cattle efficiency can decrease production costs. Similarly, improvement of carcass traits has become a highlight of interest as premiums for high-quality carcasses drives selection decisions. With the rapid increase in the world population and a decrease in available resources, the focus on economically relevant (i.e., growth and carcass quality) traits has proven to aid in the financial conservation of the beef industry (Carstens and Tedeschi, 2006).

Currently, very little research has been conducted to evaluate the genetic relationship between PAP and both feedlot and carcass performance. Boldt et al., (2014) discussed the phenotypic relationship between PAP with average daily gain (ADG) and average dry matter intake (ADMI). Additionally, a study evaluating the genetic relationship between PAP with ultrasound traits (Pauling, 2017). Because of this lack of research, little is known regarding the genetic antagonisms that could potentially exist between these traits. Many producers in areas of high elevation have continuously selected against high PAP in their herds. With the American Angus Association developing a PAP EPD and many other breed associations developing similar genetic evaluations, it is essential to understand the potential antagonisms between PAP and economically relevant traits. Therefore, the objective of this research was to evaluate the genetic relationship between PAP and feedlot and post-harvest performance traits.

MATERIALS AND METHODS

The Institutional Animal Care and Use Committee at Colorado State University (number 17-7179A) approved all animal procedures.

Animal information and data collection:

Cattle used for this study were from the Colorado State University Beef Improvement Center (CSU-BIC) spring-calving Angus herd. The CSU-BIC is located near Encampment, WY, and is at an approximate elevation of 2,115 m. From this herd, 6,898 animals were PAP tested from the years 1993 to 2017. The average starting age of testing was 384 ± 80 days, with a range of 223 to 543 days. After weaning, calves not chosen for development and purchase in the CSU-BIC annual bull test and sale were castrated and sent to the Eastern Colorado Research Center (ECRC), a moderate elevation (1,420 m) feedlot for finishing. Cattle were then harvested in facilities located in Colorado and Wyoming and carcass data were collected on 1,627 steers from the years 2001 to 2018, with an average harvest age of 578 ± 87.89 days.

From this research herd, individual feed intake measurements were obtained on 558 steers born in the years 2013 to 2017. Steers were placed in the Colorado State University Feed Intake Unit (CSU-FIU; elevation 1,557 m) to collect individual feed intake measurements using the Growsafe Monitoring Systems®. Upon arrival at the testing facility, cattle were processed and sorted into group pens based on starting weight. Test starting age and length of test varied by year and is further detailed in Table 3.1. All steers were given a 21-day warm-up period to adapt to the testing facilities and diet. The diet provided for the feed intake test was consistent throughout all five years of testing and is further detailed in Table 3.2. Approximately every 14 days, cattle were weighed using a scale attached to a squeeze chute to obtain an average daily gain (ADG) measurement.

Table 3.1: Feed intake test summary

Test year	Length of test in days	Number of steers	Average Starting age in days ± SD	Arrival Date
2014	70	93	490 ± 23.47	8/14/2014
2015	77	111	455 ± 27.27	7/16/2015
2016	69	110	377 ± 24.09	4/13/2016
2017	74	97	376 ± 20.94	4/14/2017
2018	73	147	278 ± 24.69	1/16/2018

Table 3.2: Feed ration utilized for intake study

Ingredient	Ration %
Corn Silage	10%
Alfalfa hay	6.90%
Cracked corn	74.46%
Dry distillers grain	3.79%
Limestone	0.75%
Mineral Supplement	4.10%

Statistical Analysis:

Heritabilities and genetic correlations were obtained using the statistical software package ASReml 3.0 (Gilmour et al., 2009). For these analyses, a series of 5-trait models were implemented to evaluate the potential genetic relationship between PAP, feedlot, and carcass traits. For each model, PAP, ADG, average dry matter intake (ADMI), and weaning weight (WW) were included and a single carcass trait was rotated throughout the series. In this study, the carcass quantity/quality traits evaluated were ribeye area (REA), marbling score (MARB), back fat (BF), hot carcass weight (HCW), and calculated yield grade (CYG).

For the purpose of variance component estimation, a 3-generation ancestral pedigree was constructed from the final data set. This pedigree was used for each of the 5-trait analysis and contained 12,699 individual animals with 348 unique sires and 1,904 unique dams. The inbreeding levels ranged from 0% to 26%, with an average inbreeding coefficient of 1.6%.

To conduct these analyses, fixed effects for PAP included PAP contemporary group (sex and PAP date) and PAP age as a linear covariate. For feedlot performance traits, fixed effects included test length, feed intake test contemporary group (weaning date and intake test pen) and starting age as a linear covariate. Each carcass trait included the fixed effects of feed intake test contemporary group, slaughter date, and slaughter age as a linear covariate. For PAP, feedlot, and carcass traits, individual animal was the sole direct, additive genetic random effect. Fixed effects for WW included WW contemporary group (weaning date), Beef Improvement Federation (BIF) age of dam classification, sex, and weaning age as a linear covariate as well as direct and maternal additive genetic; and permanent environmental random effects (BIF, 2018). Table 3.3 visually describes the fixed and random effects included for each trait in this analysis.

Table 3.3: Description of fixed and random effects included in the multivariate analysis of pulmonary arterial pressure, average daily gain, average dry matter intake, ribeye area, marbling score, back fat, calculated yield grade, and hot carcass weight.

	Model			
Effect[1]	PAP	FI	CT	WW
Fixed:				
PAP Age [2]	X			
PAPCG [3]	X			
FICG [4]		X	X	
Test Length		X		
Test Age [2]		X		
Kill Age [2]			X	
Kill Date			X	
WCG [5]				X
Weaning Age [2]				X
Sex				X
Age of dam				X
Random:				
Animal	X	X	X	X
Maternal				X
Maternal Permanent Environment				X

[1] PAP = Pulmonary arterial pressure, FI = Feed intake trait, CT = Carcass traits, WW = Weaning weight

[2] Linear covariate

[3] PAPCG = PAP contemporary group (sex, PAP date, and PAP score)

[4] FICG = Intake test contemporary group (weaning date and intake test pen)

[5] WCG = Weaning contemporary group (weaning date)

The following is the general form of the mixed model equations in matrix notation used in the analysis:

$$\begin{bmatrix} y_1 \\ y_2 \\ y_3 \\ y_4 \\ y_5 \end{bmatrix} = \begin{bmatrix} X_1 & 0 & 0 & 0 & 0 \\ 0 & X_4 & 0 & 0 & 0 \\ 0 & 0 & X_3 & 0 & 0 \\ 0 & 0 & 0 & X_4 & 0 \\ 0 & 0 & 0 & 0 & X_5 \end{bmatrix} \begin{bmatrix} b_1 \\ b_2 \\ b_3 \\ b_4 \\ b_5 \end{bmatrix} + \begin{bmatrix} Z_{a_1} & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & Z_{a_2} & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & Z_{a_3} & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & Z_{a_4} & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & Z_{a_5} & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & Z_{m_5} & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & Z_{p_5} \end{bmatrix} \begin{bmatrix} u_{a_1} \\ u_{a_2} \\ u_{a_3} \\ u_{a_4} \\ u_{a_5} \\ u_{m_5} \\ u_{p_5} \end{bmatrix}$$

$$+ \begin{bmatrix} e_1 \\ e_2 \\ e_3 \\ e_4 \\ e_5 \end{bmatrix},$$

where y_i was a vector of observations for the *ith* trait, X_i was an incidence matrix relating unknown fixed effect solutions in $\mathbf{b}_i$ to observations in y_i, Z_{ai} was an incidence matrix relating unknown additive (a) random effect solutions in $\mathbf{u}_{ai}$ to observations in y_i, Z_{m5} was an incidence matrix relating unknown maternal (m) random genetic effect solutions in $\mathbf{u}_{m5}$ to observations in y_i, Z_{p5} was an incidence matrix relating unknown maternal permanent environmental (p) random additive effects solutions in $\mathbf{u}_{p5}$ to observations in y_i, and e_i was a vector of random residual errors for each record.

With (co)variances equal to:

$$Var\begin{bmatrix} u_{a1} \\ u_{a2} \\ u_{a3} \\ u_{a4} \\ u_{a5} \\ u_{m5} \end{bmatrix} = \begin{bmatrix} \sigma_{a1}^2 & \sigma_{a1a2} & \sigma_{a1a3} & \sigma_{a1a4} & \sigma_{a1a5} & \sigma_{a1m5} \\ \sigma_{a2a1} & \sigma_{a2}^2 & \sigma_{a2a3} & \sigma_{a2a4} & \sigma_{a2a5} & \sigma_{a2m5} \\ \sigma_{a3a1} & \sigma_{a3a2} & \sigma_{a3}^2 & \sigma_{a3a4} & \sigma_{a3a5} & \sigma_{a3m5} \\ \sigma_{a4a1} & \sigma_{a4a3} & \sigma_{a3a4} & \sigma_{a4}^2 & \sigma_{a4a5} & \sigma_{a4m5} \\ \sigma_{a5a1} & \sigma_{a5a2} & \sigma_{a5a3} & \sigma_{a5a4} & \sigma_{a5}^2 & \sigma_{a5m5} \\ \sigma_{m5a1} & \sigma_{m5a2} & \sigma_{m5a3} & \sigma_{m5a4} & \sigma_{m5a5} & \sigma_{m5}^2 \end{bmatrix} \otimes A$$

maternal permanent environmental variance equal to:

$$\boldsymbol{MPE = Var\left[u_{p_5}\right] I}$$

and residual (co)variance equal to:

$$\boldsymbol{Var\begin{bmatrix} e_1 \\ e_2 \\ e_3 \\ e_4 \\ e_5 \end{bmatrix} = \begin{bmatrix} \sigma^2_{e_1} & \sigma_{e_{12}} & \sigma_{e_{13}} & \sigma_{e_{14}} & \sigma_{e_{15}} \\ \sigma_{e_{21}} & \sigma^2_{e_2} & \sigma_{e_{23}} & \sigma_{e_{24}} & \sigma_{e_{25}} \\ \sigma_{e_{31}} & \sigma_{e_{32}} & \sigma^2_{e_3} & \sigma_{e_{34}} & \sigma_{e_{35}} \\ \sigma_{e_{41}} & \sigma_{e_{42}} & \sigma_{e_{43}} & \sigma^2_{e_4} & \sigma_{e_{45}} \\ \sigma_{e_{51}} & \sigma_{e_{52}} & \sigma_{e_{53}} & \sigma_{e_{54}} & \sigma^2_{e_5} \end{bmatrix} \otimes I}$$

where $\boldsymbol{A}$ was Wright's numerator relationship matrix, $\boldsymbol{\sigma^2_{ai}}$ was the direct genetic variance for trait *i*, $\boldsymbol{\sigma^2_{m5}}$ was the maternal genetic variance for trait *i*, $\boldsymbol{\sigma_{aij}}$ was the direct genetic covariance between trait *i* and *j*, $\boldsymbol{\sigma_{aim5}}$ was the covariance between the direct component of trait *i* and the maternal component of trait *i*, $\boldsymbol{\sigma^2_{ei}}$ was the residual variance for trait *i*, and $\boldsymbol{\sigma_{eij}}$ was the residual covariance of traits *i* and *j*, ⊗ was the Kronecker product operator ***I*** was an identity matrix with an order equal to the number of observations in $\boldsymbol{y_i}$ (Wright, 1922).

RESULTS AND DISCUSSION

Summary statistics for PAP, feedlot performance, carcass traits, and WW are further detailed in table 3.4. Number of observations, mean, standard deviation, and the range PAP, feedlot performance, carcass traits, and WW are presented in table 3.4. Summary statistics for all traits analyzed were within the range of previously published results (Boldt et al., 2014; Boldt et al., 2018). Genetic and residual variances from these analyses are detailed in Tables 3.5 and 3.6, respectfully. Results (heritabilities and genetic correlations) from the series of multivariate analyses for PAP, ADG, ADMI, REA, MARB, BF, HCW, CYG, and WW are summarized in Table 3.7.

Table 3.4: Summary statistics of pulmonary arterial pressure, weaning weight, average daily gain, average dry matter intake, ribeye area, marbling score, back fat, hot carcass weight, and calculated yield grade

	N	Mean	SD	Minimum	Maximum
PAP, mmHg	6898	42.2	9.6	21	139
WW, kg	9026	214.1	30.9	97.9	368.3
ADG, kg/d	558	1.5	0.3	0.3	2.4
ADMI, kg/d	558	11.5	2.3	4.3	19.2
REA, cm^2	1627	81.0	9.3	35.5	119.9
MARB	1627	585	116	90	970
BF, mm	1627	14.5	3.8	2.5	43.7
HCW, kg	1627	382.9	46.9	171.5	519.3
CYG	1499	3.6	0.6	1.5	5.0

PAP = Pulmonary arterial pressure, WW = Weaning weight, ADG = Average daily gain, ADMI = Average dry matter intake, REA = Ribeye area, MARB = Marbling score, BF = Back fat, HCW = Hot carcass weight, CYG = Calculated yield grade

Heritabilities:

Heritability estimates for PAP, ADG, ADMI, and WWT were calculated by averaging variance estimates across all 5-trait models, followed by the highest reported calculated standard error. The average heritability for PAP was reported to be 0.29 ± 0.03 and was in the range of previously reported literature (0.26 ± 0.03 to 0.46 ± 0.16; Enns et al., 1992; Crawford et al., 2016; Pauling et al., 2018). Both feedlot performance traits evaluated in this study were moderately heritable, with average estimates being 0.42 ± 0.10 (ADG) and 0.29 ± 0.11 (ADMI). Estimates were similar to other reported literature estimates for Angus cattle for both ADG (0.35 ± 0.03 to 0.41 ± 0.08; MacNeil et al., 1991; Arthur et al., 1997; Schenkel et al., 2004) and ADMI (0.39 ± 0.03 to 0.42 ± 0.13; Arthur et al., 2001; Elzo et al., 2009). The carcass traits included in this evaluation (REA, MARB, BF, HCW, and CYG) were all moderately heritable estimates, 0.28 ± 0.05, 0.27 ± 0.06, 0.27 ± 0.06, 0.43 ± 0.06, and 0.28 ± 0.06, respectfully and were within the range of previously reported results (Boldt et al., 2018; Torres-Vázquez et al., 2018).

Genetic Correlations:

Because of the multiple models used for this project, reported genetic correlations not including a carcass trait were reported as the average estimates calculated in the series of 5-trait analyses followed by the highest calculated standard error. The average genetic correlations between PAP with ADG and ADMI were 0.03 ± 0.17 and 0.32 ± 0.20, respectfully. These results are complementary to those reported in a multi-breed study that evaluated growing beef cattle and concluded that cattle with higher PAP scores also showed an increase in daily feed intake (Maddock et al., 2010). Cattle who have an increased intake spend more time visiting the feed bunks consuming more feed rather than resting, causing an increased workload of their cardiovascular system (Munro et al., 2019). Previously, it was suggested that increased growth and muscle mass could be associated with high PAP scores (Pauling, 2017). Furthermore, a study which evaluated the performance of Angus steers as they moved from a high elevation operation to a moderate elevation feedlot determined that while in the feedlot, the majority of steers with higher PAP score were larger and fatter than their low PAP score contemporaries (Neary et al., 2015). These results would suggest that cattle with higher PAP scores are traditionally less efficient at converting feed when compared to their contemporaries. Potentially, these animals could be less efficient due to poor cardiopulmonary function causing their lack of efficiency, independent from their fat content and size.

The genetic relationship between PAP and carcass traits (REA, MARB, BF, HCW, and CYG) were -0.30 ± 0.12, 0.01 ± 0.13, -0.07 ± 0.13, 0.15 ± 0.10, and 0.29 ± 0.12, respectively. Similar findings were reported from a study that evaluated the genetic relationship between PAP and ultrasound carcass traits of Angus cattle with the results of REA, intramuscular fat (IMF), and BF were 0.24 ± 0.12, -0.04 ± 0.10, and -0.03 ± 0.12, respectfully (Pauling, 2017). The negative correlation between PAP and REA, BF, and HCW, suggests that the presence of PH is not an issue that directly related to fatter larger framed cattle. These results suggest that cattle with high PAP could be less efficient, causing marginal carcass quality when fed out at moderate elevation.

Moreover, these results suggest that continuous selection against high PAP cattle at high elevations will not drive adverse influences on postharvest performance when finished at moderate elevation. The genetic relationship between PAP with weaning weight direct (WWD) and weaning weight maternal (WWM) was low but positive, with average estimates of 0.18 ± 0.10 and 0.10 ± 0.10, respectfully.

Table 3.5: Estimates of residual variance (diagonal) and covariance (lower diagonal) from the analysis of pulmonary arterial pressure, average daily gain, average dry matter intake, ribeye area, marbling score, back fat, calculated yield grade, and hot carcass weight

	PAP	ADG	ADMI	REA	MARB	BF	HCW	CYG	WW
PAP	60.27[1]	-	-	-	-	-	-	-	-
ADG	-0.44[1]	0.17[1]	-	-	-	-	-	-	-
ADMI	-3.37[1]	0.38[1]	9.01[1]	-	-	-	-	-	-
REA	-0.33	0.03	0.17	0.99	-	-	-	-	-
MARB	-13.98	-0.01	15.06	-	7539.00	-	-	-	-
BF	-0.09	0.00	0.04	-	-	0.01	-	-	-
HCW	-76.84	7.14	48.41	-	-	-	3442.00	-	-
CYG	-0.36	0.00	0.14	-	-	-	-	0.21	-
WW	-2.33[1]	1.69[1]	26.30[1]	9.90[1]	-16.74[1]	0.50[1]	1089.00[1]	2.53[1]	1541.60[1]

[1] Reported as the average variance and covariance of all 5-trait multivariate analysis

PAP = Pulmonary arterial pressure, ADG = Average daily gain, ADMI = Average dry matter intake, REA = Rib eye area, MARB = Marbling score, BF = Back fat, HCW = Hot carcass weight, CYG = Calculated yield grade, WW = Weaning weight

Table 3.6: Estimates of genetic variance (diagonal) and covariance (lower diagonal) from the analysis of pulmonary arterial pressure, average daily gain, average dry matter intake, ribeye area, marbling score, back fat, calculated yield grade, and hot carcass weight

	PAP	ADG	ADMI	REA	MARB	BF	HCW	CYG	WWD	WWM
PAP	24.87[1]	-	-	-	-	-	-	-	-	-
ADG	0.06[1]	0.12[1]	-	-	-	-	-	-	-	-
ADMI	3.11[1]	0.36[1]	3.76[1]	-	-	-	-	-	-	-
REA	-0.92	0.13	0.38	0.39	-	-	-	-	-	-
MARB	1.00	5.73	3.59	-	2837.00	-	-	-	-	-
BF	-0.02	0.01	0.05	-	-	0.00	-	-	-	-
HCW	39.42	18.94	81.57	-	-	-	2642.00	-	-	-
CYG	0.42	0.05	0.27	-	-	-	-	0.08	-	-
WWD	18.50[1]	5.44[1]	13.97[1]	3.13[1]	135.00[1]	0.03[1]	555.10[1]	1.32[1]	425.14[1]	-
WWM	9.92[1]	1.55[1]	22.28[1]	1.69[1]	-105.60[1]	0.41[1]	576.90[1]	2.85[1]	-67.27[1]	391.08[1]

[1] Reported as the average variance and covariance of all 5-trait multivariate analysis

PAP = Pulmonary arterial pressure, ADG = Average daily gain, ADMI = Average dry matter intake, REA = Rib eye area, MARB = Marbling score, BF = Back fat, HCW = Hot carcass weight, CYG = Calculated yield grade, WWD = Weaning weight direct, WWM = Weaning weight maternal

Table 3.7: Heritability estimates (diagonal) and genetic correlations (above diagonal) followed by their standard error from the analysis of pulmonary arterial pressure, average daily gain, average dry matter intake, ribeye area, marbling score, back fat, calculated yield grade, and hot carcass weight

	PAP	ADG	DMI	REA	MARB	BF	HCW	CYG	WWD	WWM
PAP	0.29[1] (0.03)	0.03[1] (0.17)	0.32[1] (0.20)	-0.30 (0.12)	0.01 (0.13)	-0.07 (0.13)	0.15 (0.10)	0.29 (0.12)	0.18[1] (0.10)	0.10[1] (0.10)
ADG	-	0.42[1] (0.10)	0.52[1] (0.20)	0.60 (0.19)	0.32 (0.21)	0.38 (0.19)	0.94 (0.06)	0.57 (0.17)	0.76[1] (0.15)	0.22[1] (0.22)
DMI	-	-	0.29[1] (0.11)	0.33 (0.23)	0.04 (0.26)	0.34 0.22)	0.75 (0.12)	0.50 (0.20)	0.35[1] (0.21)	0.58[1] (0.27)
REA	-	-	-	0.28 (0.05)	-	-	-	-	0.24 (0.14)	0.14 (0.15)
MARB	-	-	-	-	0.27 (0.06)	-	-	-	0.12 (0.15)	-0.10 (0.16)
BF	-	-	-	-	-	0.27 (0.06)	-	-	0.02 (0.15)	0.30 (0.16)
HCW	-	-	-	-	-	-	0.43 (0.06)	-	0.52 (0.10)	0.56 (0.10)
CYG	-	-	-	-	-	-	-	0.28 (0.06)	0.22 (0.14)	0.50 (0.15)
WWD	-	-	-	-	-	-	-	-	0.14[1] (0.03)	-0.16[1] (0.12)
WWM	-	-	-	-	-	-	-	-	-	0.13[1] (0.03)

[1] Reported as the average estimate and largest SE of all 5-trait multivariate analyses

PAP = Pulmonary arterial pressure, ADG = Average daily gain, ADMI = Average dry matter intake, REA = Rib eye area, MARB = Marbling score, BF = Back fat, HCW=Hot carcass weight, CYG = Calculated yield grade, WWD = Weaning weight direct, WWM = Weaning weight maternal

CONCLUSIONS

In the current study evaluating high elevation cattle with their performance at moderate elevation feedlots, results suggest that cattle with high yearling PAP scores suffer from poor cardiopulmonary health, feedlot performance, and carcass quality. The moderate positive correlation between PAP and ADMI suggested that cattle with higher yearling PAP scores consumed more feed, potentially leading to a decrease in feed efficiency. Additionally, the reported genetic relationships between PAP and carcass traits (REA, BF, MARB, HCW, and CYG) prelude that cattle with lower yearling PAP scores develop heavier muscled learner carcasses in contrast to their high PAP score contemporaries. High PAP cattle who are transported from high to moderate elevations could potentially be using excess energy towards their pulmonary cardiovascular system and possibly suffering from cardiac cachexia resulting in a decrease in feed efficiency and a reduction in carcass quality.

IMPLICATIONS

High mountain disease, often referred to as brisket disease, has been observed in beef cattle operations in the mountain regions for at least a century. Continuous phenotypic selection using PAP has shown to develop cattle that are better adapted to these high elevation regions decreasing their risk of developing PH. Results from this analysis suggested that continuous selection against high PAP cattle will not negatively impact the subsequent generation's feedlot and post-harvest performance. Undesirably, cattle who are culled from operations because of high PAP scores, have the potential for poor feedlot performance with a lighter muscled carcass, if fed at moderate elevations. As economic popularity in the beef industry is driven by cattle efficiency and adaptability, this information can aid producers at high elevation as they select traits to include to their breeding objectives. These findings indicate that feedlots can purchase cattle from high elevation producers with selection pressure for PAP in their herd without subsequently having undesirable feedlot and post-harvest performance.

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CHAPTER 4

DATA AND ANALYSIS INFORMATION; MULTIVARIATE ANALYSIS OF PULMONARY ARTERIAL PRESSURE, FEEDLOT PERFORMANCE, AND CARCASS TRAITS

INTRODUCTION

In areas of high elevation, hypoxia-induced pulmonary hypertension (PH) causes right ventricle failure and can lead to the manifestation of high altitude disease (HAD), often called brisket disease. Limited oxygen in areas of high elevation causes vasoconstriction in pulmonary vasculature (Farber and Loscalzo, 2004). Holt and Callan, (2007) reported that approximately 5% death loss for cattle who reside in high elevation regions could be attributed to PH. Fortunately, pulmonary arterial pressure (PAP) can be used as an indicator trait to identify cattle who have the potential to develop (PH). Neary et al., (2015) suggested that as cattle enter feedlot systems, an increase in adipose tissue and ruminal engorgement could increase PAP. Rapid weight gain and high intake of concentrate diets for cattle in feedlots cause increased work of the cardiac ventricle and sustained accelerated circulation (Jensen et al., 1976).

Feed efficiency, a ratio of inputs to outputs, is vital in the beef industry as feed costs represent up to 70% of the total expenses of production (Shike, 2013). Also, premiums for higher quality carcasses, as well as an increase in popularity of branded beef programs has drawn interest in the genetic merit of carcass traits. As indexes of EPD are becoming more readily available for these traits, its logical that enhanced selection pressure is occurring in breeding objectives. Also, since the American Angus Association is publishing a PAP EPD, it is crucial to understand the potential antagonisms between PAP and feedlot performance and carcass quality. We hypothesized that cattle with higher yearling PAP scores at high elevation would show a general decrease in feedlot performance and an increase in fat-related carcass traits when finished at moderate elevations. Therefore, the objective of this study was to determine if a genetic relationship exists between PAP and feedlot performance and carcass traits.

MATERIALS AND METHODS

The Institutional Animal Care and Use Committee at Colorado State University (number 17-7179A) approved all animal procedures

Cattle Information:

Cattle used for this study were from research herd at the Colorado State University Beef Improvement Center (CSU-BIC) located near Saratoga, WY (elevation 2,340 m). This operation has a spring calving Angus herd with 420 cows on 2597 ha. Primary forage for this herd contains hay harvested onsite and improved native pasture grazing. Each year, female progeny are selected to keep as replacement heifers. Additionally, male progeny who have undergone genetic selection based on phenotypic values and expected progeny difference values for industry-related traits (i.e. growth and birth weight) can be selected to go through a bull development program and are offered in an annual sale. Calves that meet the qualifications stated above are chosen to enter the bull performance test while the remaining male calves are castrated and sent to a moderate elevation feedlot (elevation 1,420 m).

PAP:

This spring calving herd has undergone selection pressure against high pulmonary arterial pressure (PAP) for 20 years. Table 4.1 describes the number of PAP observations by year. As PAP collection is a cute side procedure, a licensed veterinarian collected PAP scores at the CSU-BIC. Information on PAP collection is further detailed by (Holt and Callan, 2007). From this operation, a total of 6,898 PAP observations have been collected, with an average collection age for PAP score being 339 ± 43 days with a range of 166 to 450 days. Additional information of PAP characteristics for this analysis is provided in Table 4.2. The distribution of PAP is visualized in Figure 4.1. Similar to previously published efforts, the distribution of PAP violates the assumption of normality as represented by skewed right-tailed distribution (Zeng, 2016; Pauling, 2017; Cockrum et al., 2019). For this analysis, PAP was included in its raw form (non-transformed). Though this violates the assumption of normality, previous studies have suggested that the transformation of PAP data may not be necessary for the calculation of heritabilities and genetic correlations as it increases the complexity for translating the results (Zeng, 2016; Speidel et al., 2020).

Table 4.1: Number of pulmonary arterial pressure observations from the Colorado State University Beef Improvement Center Angus herd by Year

Year	n	Year	n
1992	10	2006	177
1993	8	2007	230
1994	400	2008	327
1995	41	2009	287
1996	243	2010	249
1997	253	2011	469
1998	242	2012	293
1999	274	2013	375
2000	409	2014	390
2001	283	2015	434
2002	188	2016	264
2003	189	2017	398
2004	180	2018	398
2005	108		

Table 4.2: Summary statistics of pulmonary arterial pressure (PAP) for animals from the Colorado State Beef Improvement Center

	N	Mean	SD	Minimum	Maximum
PAP, mmHg	6898	42.28	9.61	21	139

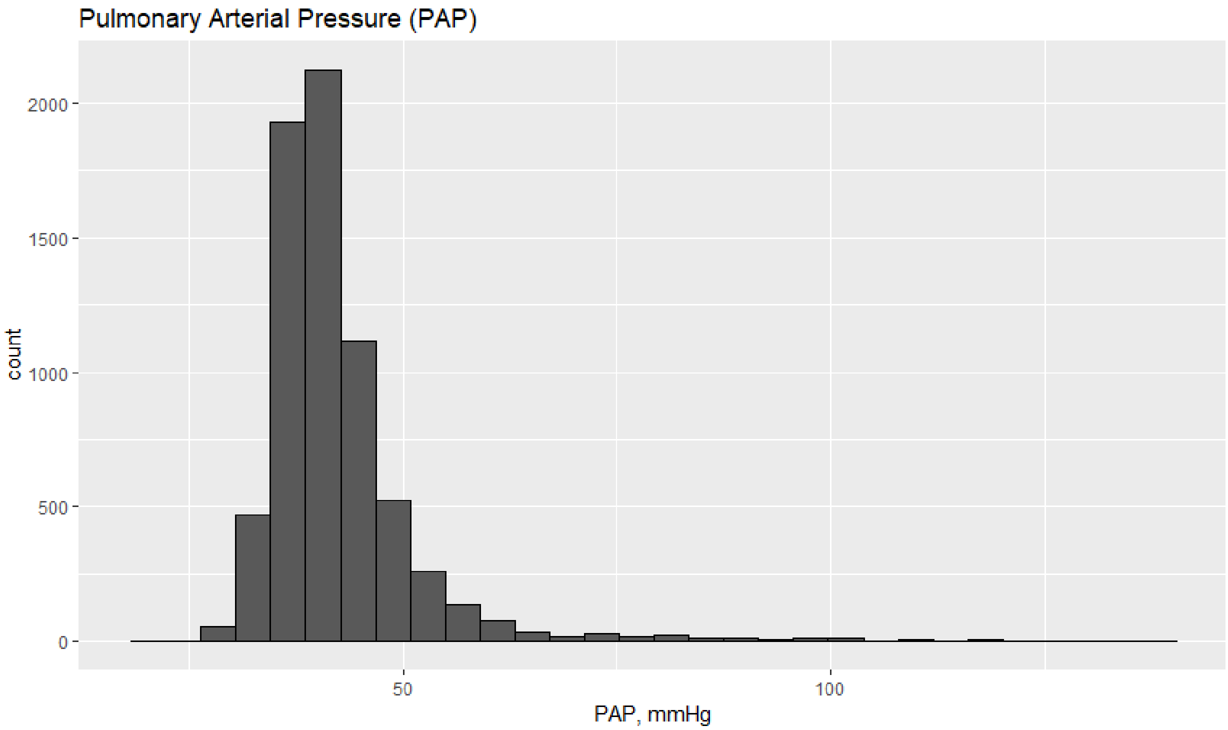


Figure 4.1: Distribution of pulmonary arterial pressure observations from the Colorado State University Beef Improvement Center Angus herd (n = 6,898; years 1992-2018)

Feed intake data:

Using steers from the CSU-BIC (n = 558), feedlot performance data was collected from the years 2012 to 2018 to be included in this analysis. Summary Statistics for feedlot performance observations are provided in Table 4.3. Steers entered the Colorado State University Feed Intake Unit (CSU-FIU) located in Fort Collins, CO (elevation 1,557m; Figure 4.2), and spent approximately 70 days in the testing facilities (Table 4.4). Individual feed intake data was collected using the Growsafe® monitoring systems, which provided daily intake values used to calculate average dry matter intake (ADMI). Cattle were sorted by weight upon arrival and placed into group pens. All were given a 21d warm-up period to adapt to testing facilities and diet. The test diet was consistent through all five years of testing and is further detailed in Table 4.5. Cattle were weighed every two weeks to obtain average daily gain (ADG) observations.

Table 4.3: Summary statistics of average daily gain (ADG) and average dry matter intake (ADMI) for animals from the Colorado State Beef Improvement Center

	N	Mean	SD	Minimum	Maximum
ADG, kg/d	558	1.656	0.286	0.30	2.44
ADMI, kg/d	558	11.49	2.33	4.34	19.20

Figure 4.2: Colorado State University Feed Intake Unit located in Fort Collins, CO

Table 4.4: Feed intake test summary

Test year	Length of test in days	Number of steers	Average Starting age in days ± SD	Arrival Date
2014	70	93	490 ± 23.47	8/14/2014
2015	77	111	455 ± 27.27	7/16/2015
2016	69	110	377 ± 24.09	4/13/2016
2017	74	97	376 ± 20.94	4/14/2017
2018	73	147	278 ± 24.69	1/16/2018

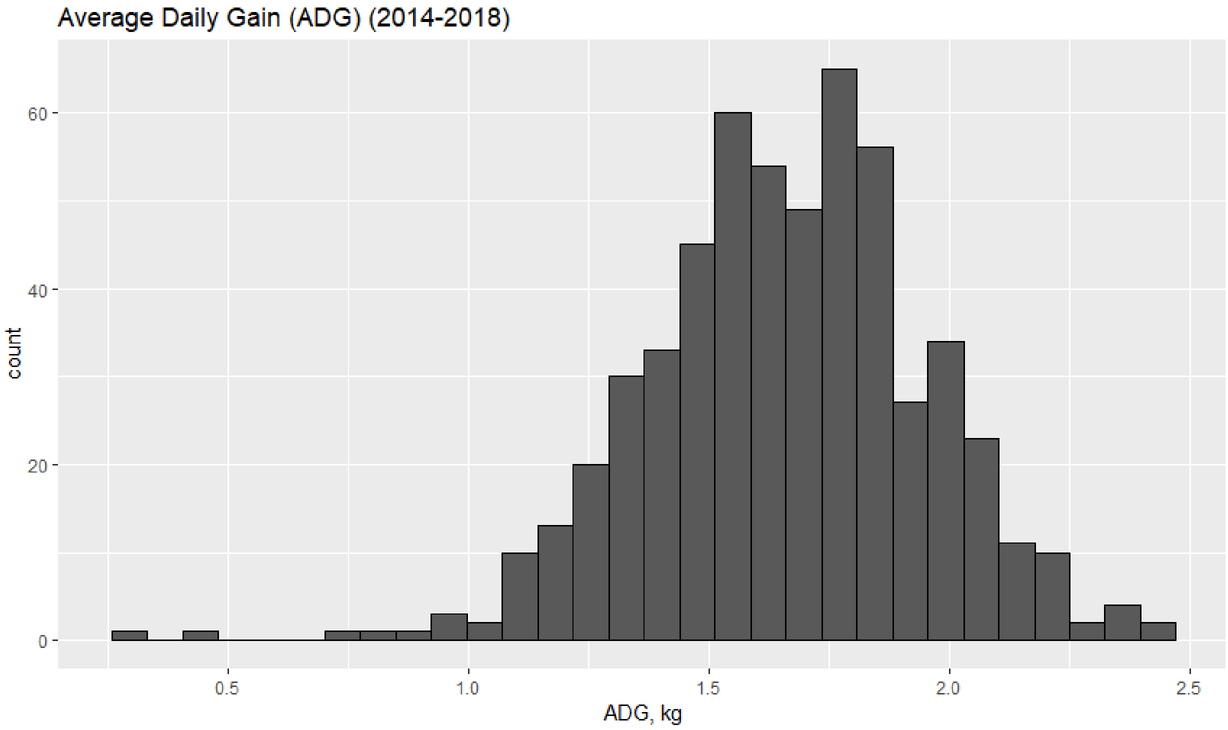
Table 4.5: Feed ration utilized for intake study

Ingredient	Ration %
Corn Silage	10%
Alfalfa hay	6.90%
Cracked corn	74.46%
Dry distillers grain	3.79%
Limestone	0.75%
Mineral Supplement	4.10%

At the beginning stages of this analysis, bivariate analyses between PAP and feedlot performance traits (ADG and ADMI) were conducted to calculate both the heritabilities and the genetic correlations for this group of steers. Surprisingly, in the first run, estimates for both heritability and genetic correlations between traits were estimated to be zero. To investigate these unexpected results further, data was subsetted by testing year to analyze individually. Through this process, it was revealed that two years of feed intake data (2012 and 2013; n = 189) were driving the estimate to zero while the remain years (2014-2018) reported estimates that were within previously reported results. Potentially, this could be a result of the inconsistent management at the CSU-FIU during these testing years.

A Shapiro-Wilks test was used to test the normality for feedlot both performance traits. Frequency distributions for ADG and ADMI (2014-2018; n = 558) are shown in Figure 4.3 For the observations included in the analysis, a non-normal distribution ($P < 0.05$) for ADG as it is skewed to the left, leaving a thin tail. In comparison, ADMI followed a normal distributed (P = 0.95). For the feedlot performance traits, there were some data extremes, showing deviations from the distribution curve, but they were analyzed and recalculated to ensure accuracy and considered biologically significant, and therefore remained in the study.

Figure 4.4 details the distribution for the feed intake data points that were removed from the analysis (n = 189; 2012-2013). For ADG, the distribution varied drastically between these data subsets (2013-2013 vs. 2014-2018) and followed a normal distribution (P = 0.25). Average dry matter intake does not follow a normal distribution (P < 0.05).



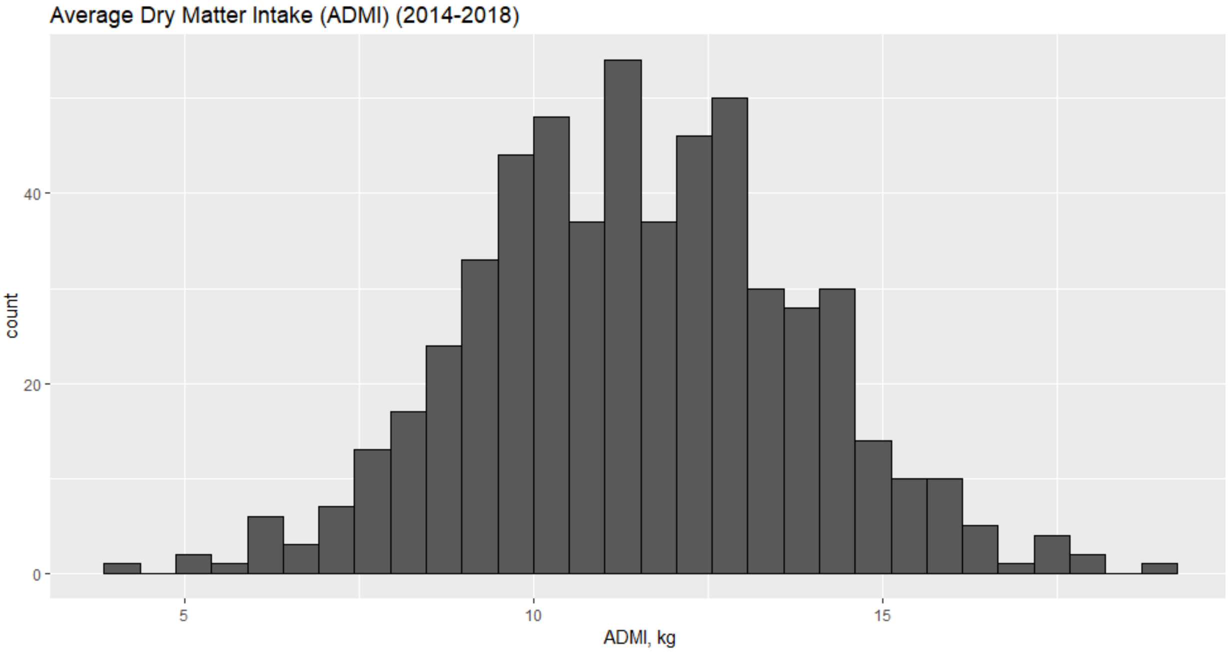
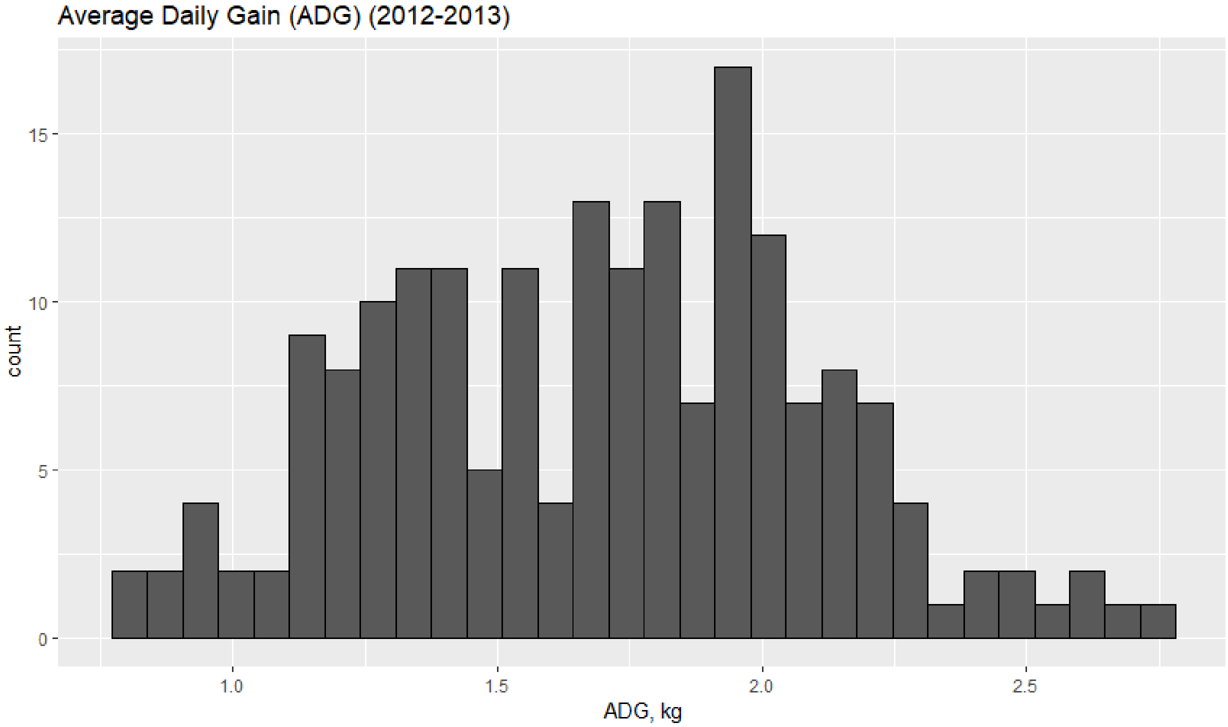


Figure 4.3: Distribution of the feedlot performance traits average daily gain (ADG) and average dry matter intake (ADMI) from the Colorado State University Beef Improvement Center steers (n = 558; years 2014-2018)



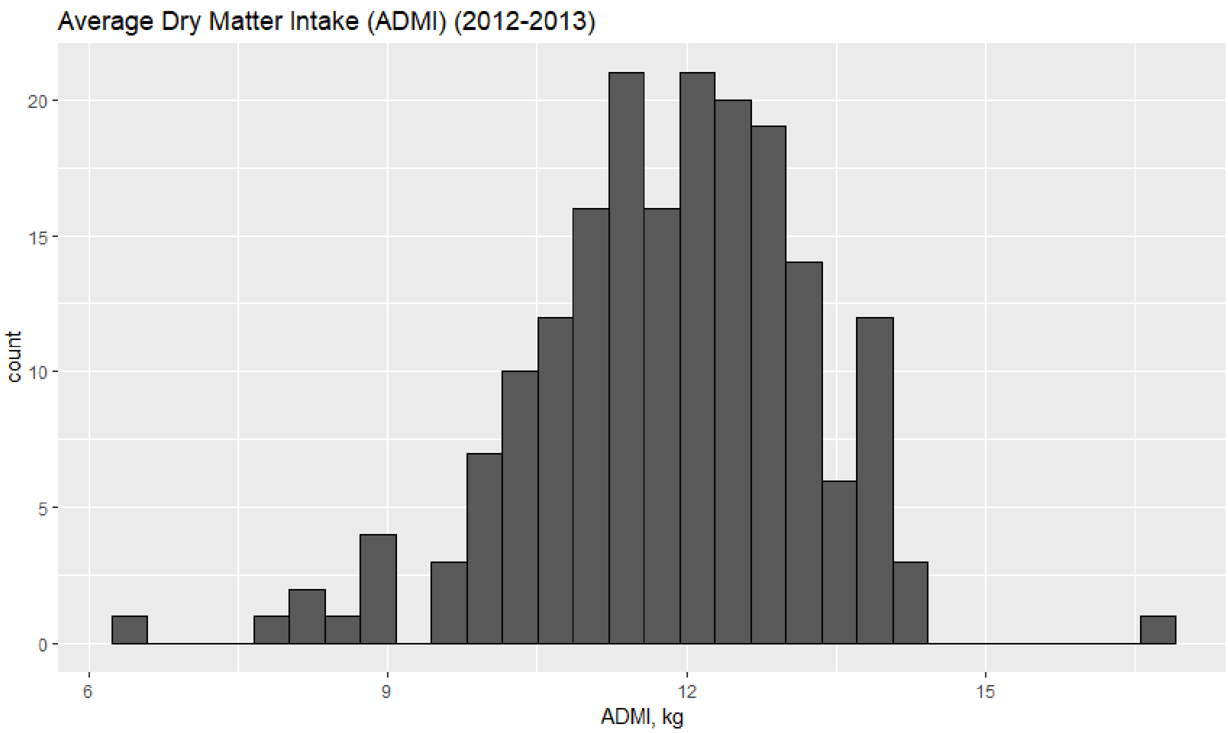


Figure 4.4: Distribution of the feedlot performance traits average daily gain (ADG) and average dry matter intake (ADMI) from the Colorado State University Beef Improvement Center steers (n = 189; years 2012-2013)

Carcass Data:

Carcass traits were collected from 1,627 steers with an average harvest age of 578 ± 87 days spanning the years 2001 to 2018. Of these steers, 482 took part in the feed intake collection at the CSU-FIU. Carcass data were provided by the facility from which the cattle were harvested, and summary statistics for carcass traits are provided in Table 4.6. Not all cattle were harvested in the same facility, but all facilities were within the states of Colorado and Wyoming.

Table 4.6: Summary statistics of ribeye area (REA), marbling score (MARB), back fat (BF), hot carcass weight (HCW), and calculated yield grade (CYG) for animals from the Colorado State Beef Improvement Center

	N	Mean	SD	Minimum	Maximum
REA, cm^2	1627	80.97	9.29	35.48	119.99
MARB	1627	585.53	116.74	90.00	970.00
BF, mm	1627	14.478	3.81	2.54	43.68
HCW, kg	1627	382.99	46.93	171.46	519.36
CYG	1499	3.55	0.56	1.50	5.00

Calculated yield grade (CYG) is an indication of the cutability of a carcass, and the formula is as follows (BIF; 2018).

$$\begin{aligned} Yield\ Grade &= 2.50 + (2.5 \times Adj. fat\ thickness, in.) \\ &+ (0.2 \times Kidney, pelvic, and\ heart\ fat, \%) \\ &+ (0.0038 \times Hot\ carcass\ wt., lb.) - (0.32 \times Ribeye\ area, sq. in.) \end{aligned}$$

In some cases, CYG was calculated and reported with a value of over 5. This is most common when cattle produce an extremely fat but light muscled carcasses. If this was the case, CYG was set as a missing observation with the remaining carcass traits for that animal being unaltered; this resulted in six CYG observations set to zero (Table 4.7). Below is the calculated yield grade formula (BIF, 2018).

Table 4.7: Detailed information regarding the six calculated yield grade values were over 5

HCW, kg	MARB	BF, mm	KPH,	REA, cm^2	CYG
319.32	580	23.37	1.75	22.24	5.12
476.73	530	20.32	2	28.19	5.342
474.00	720	24.38	2	33.78	5.01
519.36	530	23.37	2	32.51	5.46
399.16	490	26.41	2	29.71	5.10
402.79	530	43.67	2	27.94	7.05

HCW = Hot carcass weight, MARB = Marbling, BF = Back fat, KPH = Percent kidney pelvic heart fat, REA = Rib eye area, CYG = Calculated yield grade

Marbling score was converted to a numeric value following the recommendation in the Beef Improvement Federation (BIF) guidelines (Table 4.8; BIF,2018)

Table 4.8: Beef Improvement Federation (BIF) phenotypic numeric marbling scores guidelines (BIF, 2018)

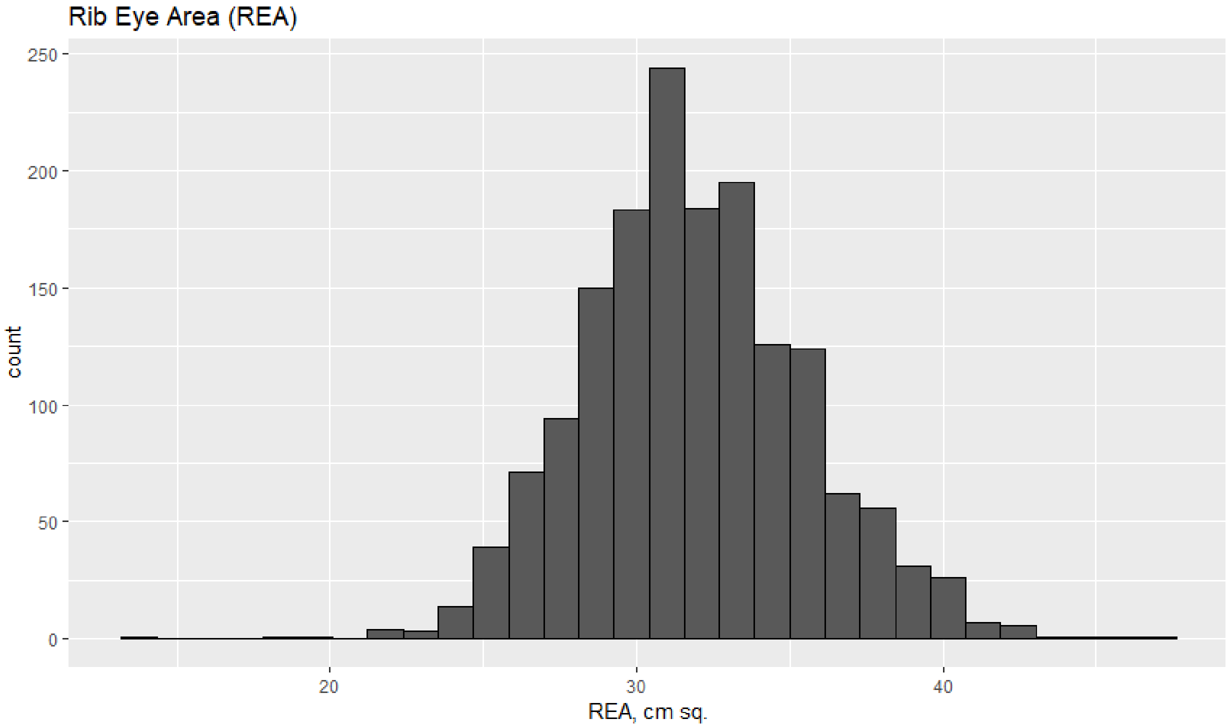
Quality Grade	Marbling	Score
Prime	Abundant	10.0 – 10.9
Prime	Moderately Abundant	9.0 – 9.9
Prime	Slightly Abundant	8.0 – 8.9
Choice	Moderate	7.0 – 7.9
Choice	Modest	6.0 – 6.9
Choice	Small	5.0 – 5.9
Select	Slight	4.0 – 4.9
Standard	Traces	3.0 – 3.9
Standard	Practically devoid	2.0 – 2.9

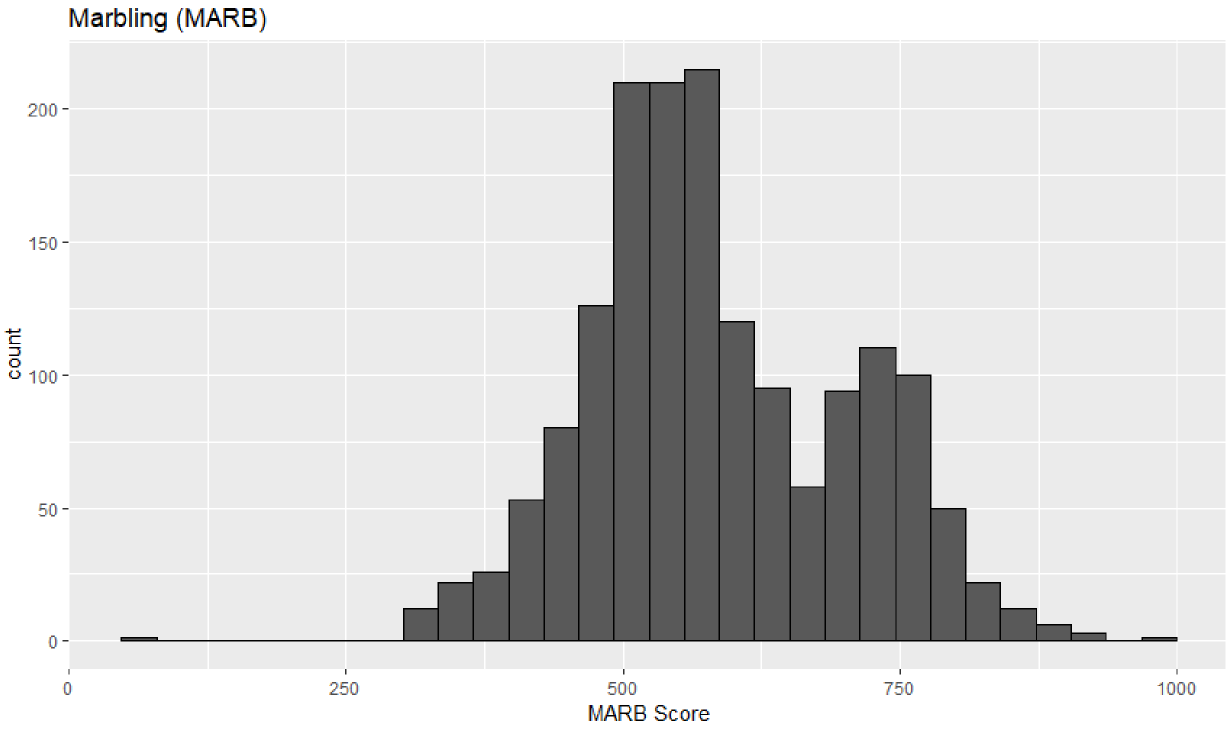
Each degree of marbling was placed into units of 100, but typically marbling scores are given in tenths within each degree of marbling (e.g., Slight ^{90}, Small ^{20}, Abundant ^{10}). Table 4.9 describes the translation of marbling scores converted to numeric values represented in the data set.

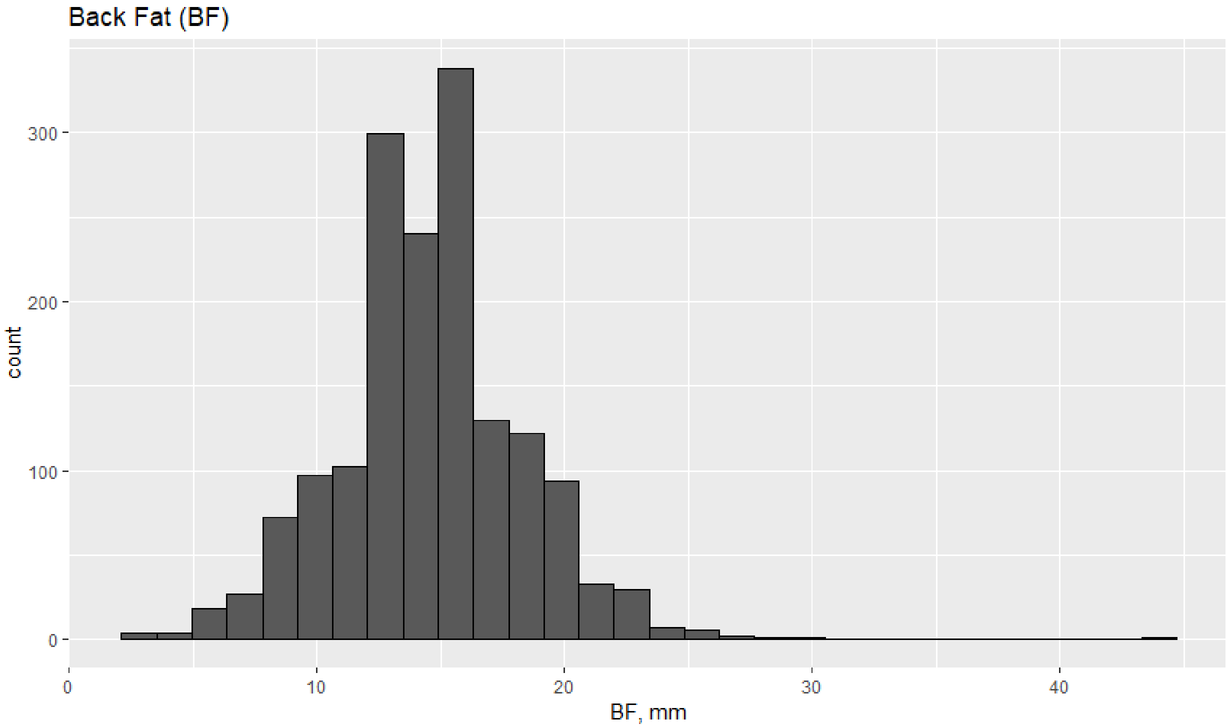
Table 4.9: Converting quality grade and marbling score into numeric units

Quality grade	Marbling score	Numeric marbling score
Choice-	Small ^{40}	540
Select	Slight ^{90}	490
Prime	Moderately Abundant ^{30}	930

The distribution of carcass data is detailed in Figure 4.5. In order to test for normality, the Shapiro-Wilks test was conducted for all carcass traits. Majority of the distributions for carcass traits showed kurtosis to some level, being that the majority of the observations seemed to fall in the middle and very few in the tails. In contrast, marbling score exhibited a bimodal distribution with the majority of marbling scores being between 500 and 600 and a smaller cluster between 650 and 730. Every carcass trait included in this analysis were considered to be non-normally distributed (REA; $p < 0.05$, MARB; $p < 0.05$, BF; $p < 0.05$, HCW; $p < 0.05$, CYG; $p < 0.05$).




Marbling (MARB)
count
200
150
100
50
0
0
250
500
750
1000
MARB Score


Back Fat (BF)
count
300
200
100
0
0
10
20
30
40
BF, mm

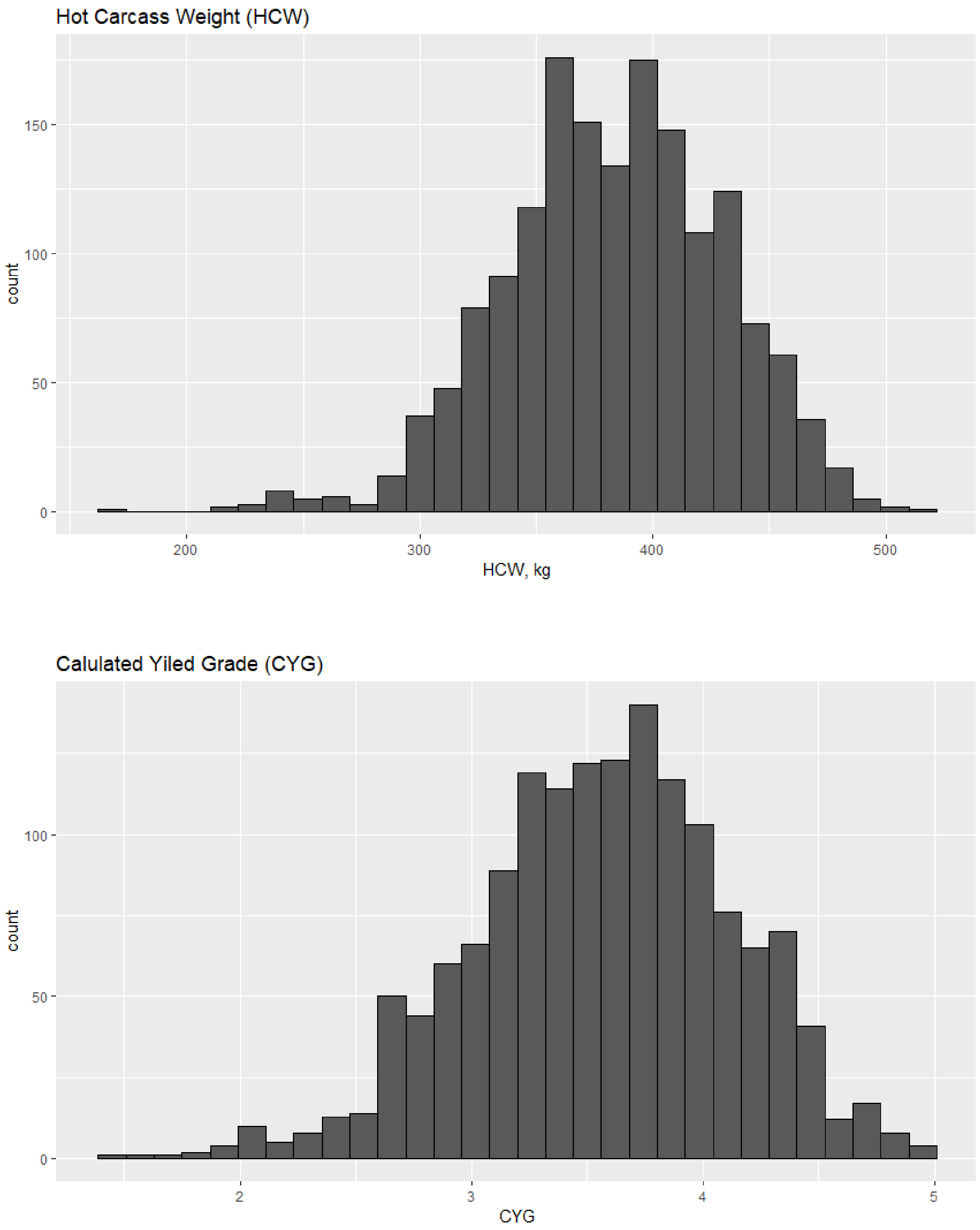


Figure 4.5: Distributions of carcass data (rib eye area, marbling score, back fat, hot carcass weight, and calculated yield grade) from the Colorado State University Beef Improvement Center Angus steers (n = 1,627)

Weaning weight data:

Summary statistics for weaning weight are detailed in Table 4.10. Weaning weight (WW) observations under 97.98 kg were individually evaluated to investigate their validity. If the ADG between weaning weight and yearling weight was greater than 1.58 kg and if WW was below 122.47 kg (3 SD below the mean), then those WW observations were removed from the data set. Frequency distribution of WW for both male and female are shown in (Figure 4.6). Based on visual inspection, these two distributions measures follow normal distributions with the majority of observations being between 190 and 240 kg.

Table 4.10: Summary statistics of weaning weight (WW) for animals from the Colorado State Beef Improvement Center

	N	Mean	SD	Minimum	Maximum
WW, kg Female and Male	9026	214.08	30.90	97.98	368.32
WW, kg Female	4340	207.13	28.32	97.98	286.67
WW, kg Male	4686	220.52	31.77	97.98	368.32

Figure 4.6: Distribution of weaning weight for both males (n = 4686) and females (n= 4340) from the Colorado State University Beef Improvement Center

Statistical Analysis:

Heritabilities and genetic correlations were obtained using the software package ASReml 3.0 (Gilmour et al., 2009). For this study, five different 5-trait models were analyzed to estimate the genetic relationship between PAP, feedlot performance, and carcass quality. In each model, traits of PAP, ADG, ADMI, and WW were included in the mixed model with a single carcass trait alternating through the series. The carcass traits included in this analysis were REA, MARB, BF, HWC, and CYG.

To calculate these variance components, an ancestral pedigree (3-generation) was created. This pedigree contained 12,699 individual animals with 348 unique sires and 1,904 unique dams with an average inbreeding coefficient of 1.6% and a range of 0% to 26%. This pedigree was used for every model ran in this analysis.

Description of the model used for this analysis is detailed in Table 4.11. In the series of models, the fixed effects for PAP included PAP contemporary group (sex and PAP date) and PAP age as a linear covariate. For feedlot performance traits, fixed effects included test length, feed intake test contemporary group (weaning date and intake test pen) and starting age as a linear covariate. Each carcass trait included the fixed effects of feed intake test contemporary group, slaughter date, and slaughter age as a linear covariate. For PAP, feedlot traits, and carcass traits, individual animal was the sole random effect which was included for the direct additive genetic effect. Fixed effects for WW included WW contemporary group (weaning date), Beef Improvement Federation (BIF) adjusted age of dam, sex, and weaning age as a linear covariate as well as direct additive, maternal, and maternal permanent environmental random effects (BIF, 2018).

Table 4.11: Description of fixed and random effects included in the multivariate analysis of pulmonary arterial pressure, average daily gain, average dry matter intake, ribeye area, marbling score, back fat, calculated yield grade, and hot carcass weight.

	Model			
Effect[1]	PAP	FI	CT	WW
Fixed:				
PAP Age [2]	X			
PAPCG [3]	X			
FICG [4]		X	X	
Test Length		X		
Test Age [2]		X		
Kill Age [2]			X	
Kill Date			X	
WCG [5]				X
Weaning Age [2]				X
Sex				X
Age of dam				X
Random:				
Animal	X	X	X	X
Maternal				X
Maternal Permanent Environment				X

[1] PAP = Pulmonary arterial pressure, FI = Feed intake trait, CT = Carcass traits, WW = Weaning weight

[2] Linear covariate

[3] PAPCG = PAP contemporary group (sex, PAP date, and PAP score)

[4] FICG = Intake test contemporary group (weaning date and intake test pen)

[5] WCG = Weaning contemporary group (weaning date)

The following is the general matrix form for the equation used for the analysis:

$$\begin{bmatrix} y_1 \\ y_2 \\ y_3 \\ y_4 \\ y_5 \end{bmatrix} = \begin{bmatrix} X_1 & 0 & 0 & 0 & 0 \\ 0 & X_4 & 0 & 0 & 0 \\ 0 & 0 & X_3 & 0 & 0 \\ 0 & 0 & 0 & X_4 & 0 \\ 0 & 0 & 0 & 0 & X_5 \end{bmatrix} \begin{bmatrix} b_1 \\ b_2 \\ b_3 \\ b_4 \\ b_5 \end{bmatrix} + \begin{bmatrix} Z_{a_1} & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & Z_{a_2} & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & Z_{a_3} & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & Z_{a_4} & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & Z_{a_5} & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & Z_{m_5} & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & Z_{p_5} \end{bmatrix} \begin{bmatrix} u_{a_1} \\ u_{a_2} \\ u_{a_3} \\ u_{a_4} \\ u_{a_5} \\ u_{m_5} \\ u_{p_5} \end{bmatrix}$$

$$+ \begin{bmatrix} e_1 \\ e_2 \\ e_3 \\ e_4 \\ e_5 \end{bmatrix},$$

where y_i was a vector of observations for the *ith* trait, X_i was an incidence matrix relating unknown fixed effect solutions in $\mathbf{b}_i$ to observations in y_i, Z_{ai} was an incidence matrix relating unknown additive (a) random effect solutions in $\mathbf{u}_{ai}$ to observations in y_i, Z_{m5} was an incidence matrix relating unknown maternal (m) random genetic effect solutions in $\mathbf{u}_{m5}$ to observations in y_i, Z_{p5} was an incidence matrix relating unknown maternal permanent environmental (p) random additive effects solutions in $\mathbf{u}_{p5}$ to observations in y_i, and e_i was a vector of random residual errors for each record.

With (co)variances equal to:

$$Var \begin{bmatrix} u_{a1} \\ u_{a2} \\ u_{a3} \\ u_{a4} \\ u_{a5} \\ u_{m5} \end{bmatrix} = \begin{bmatrix} \sigma^2_{a1} & \sigma_{a1a2} & \sigma_{a1a3} & \sigma_{a1a4} & \sigma_{a1a5} & \sigma_{a1m5} \\ \sigma_{a2a1} & \sigma^2_{a2} & \sigma_{a2a3} & \sigma_{a2a4} & \sigma_{a2a5} & \sigma_{a2m5} \\ \sigma_{a3a1} & \sigma_{a3a2} & \sigma^2_{a3} & \sigma_{a3a4} & \sigma_{a3a5} & \sigma_{a3m5} \\ \sigma_{a4a1} & \sigma_{a4a3} & \sigma_{a3a4} & \sigma^2_{a4} & \sigma_{a4a5} & \sigma_{a4m5} \\ \sigma_{a5a1} & \sigma_{a5a2} & \sigma_{a5a3} & \sigma_{a5a4} & \sigma^2_{a5} & \sigma_{a5m5} \\ \sigma_{m5a1} & \sigma_{m5a2} & \sigma_{m5a3} & \sigma_{m5a4} & \sigma_{m5a5} & \sigma^2_{m5} \end{bmatrix} \otimes A$$

maternal permanent environmental variance equal to:

$$\boldsymbol{MPE} = \boldsymbol{Var}\left[\boldsymbol{u}_{\boldsymbol{p}_5}\right]\boldsymbol{I}$$

and residual (co)variance equal to:

$$\boldsymbol{Var}\begin{bmatrix} e_1 \\ e_2 \\ e_3 \\ e_4 \\ e_5 \end{bmatrix} = \begin{bmatrix} \sigma^2_{e_1} & \sigma_{e_{12}} & \sigma_{e_{13}} & \sigma_{e_{14}} & \sigma_{e_{15}} \\ \sigma_{e_{21}} & \sigma^2_{e_2} & \sigma_{e_{23}} & \sigma_{e_{24}} & \sigma_{e_{25}} \\ \sigma_{e_{31}} & \sigma_{e_{32}} & \sigma^2_{e_3} & \sigma_{e_{34}} & \sigma_{e_{35}} \\ \sigma_{e_{41}} & \sigma_{e_{42}} & \sigma_{e_{43}} & \sigma^2_{e_4} & \sigma_{e_{45}} \\ \sigma_{e_{51}} & \sigma_{e_{52}} & \sigma_{e_{53}} & \sigma_{e_{54}} & \sigma^2_{e_5} \end{bmatrix} \otimes \boldsymbol{I}$$

where $\boldsymbol{A}$ was Wright's numerator relationship matrix, $\boldsymbol{\sigma^2_{ai}}$ was the direct genetic variance for trait *i*, $\boldsymbol{\sigma^2_{m5}}$ was the maternal genetic variance for trait *i*, $\boldsymbol{\sigma_{aij}}$ was the direct genetic covariance between trait *i* and *j*, $\boldsymbol{\sigma_{aim5}}$ was the covariance between the direct component of trait *i* and the maternal component of trait *i*, $\boldsymbol{\sigma^2_{ei}}$ was the residual variance for trait *i*, and $\boldsymbol{\sigma_{eij}}$ was the residual covariance of traits *i* and *j*, ⊗ was the Kronecker product operator *I* was an identity matrix with an order equal to the number of observations in $\boldsymbol{y_i}$ (Wright, 1922).

Additional Analysis Information

When solving for starting values for the five trait models, the bivariate analysis between KPH with ADMI and WWT was not able to solve. Descriptive statistics for KPH is detailed in Table 4.12. KPH was opted out of this analysis due to its inability to solve equations when it is included. Even when traits were set to a magnifier of 10, the bivariate models were not able to converge. This could potentially be due to the lack of variation for the trait.

Table 4.12: Summary statistics of KPH for animals from the Colorado State Beef Improvement Center

	N	Mean	SD	Minimum	Maximum
KPH	1,506	2.02	0.17	0.5	4.0

RESULTS AND DISCUSSION

Summary statistics for PAP, feedlot performance, carcass traits, and WW are further detailed in table 3.4. Number of observations, mean, standard deviation, and the range PAP, feedlot performance, carcass traits, and WW are presented in table 3.4. Summary statistics for all traits analyzed were within the range of previously published results (Boldt et al., 2014; Boldt et al., 2018). Residual and genetic variances from these analyses are detailed in Tables 4.13 to 4.22, respectfully. Results (heritabilities and genetic correlations) from the series of multivariate analyses for PAP, ADG, ADMI, REA, MARB, BF, HCW, CYG, and WW are summarized in Tables 4.23 to 4.27.

Heritability

Heritability for PAP was 0.29 ± 0.03 and was consistent with previously reported literature estimates (0.26 ± 0.03 to 0.46 ± 0.16; Enns et al., 1992; Crawford et al., 2016; Pauling et al., 2018). Feedlot performance estimates were moderately heritable, with average estimates ranging from 0.38 ± 0.09 to 0.48 ± 0.08 and 0.28 ± 0.11 to 0.39 ± 0.10 for ADG and ADMI, respectfully. These estimates were similar to those previously reported in literature for both ADG (0.35 ± 0.03 to 0.41 ± 0.08; Arthur et al., 1997; Schenkel et al., 2004) and ADMI (0.39 ± 0.03 to 0.42 ± 0.13; Arthur et al., 2001; Elzo et al., 2009). The carcass traits included in this evaluation (REA, MARB, BF, HCW, and CYG) were all moderately heritable estimates, 0.28 ± 0.05, 0.27 ± 0.06, 0.27 ± 0.06, 0.43 ± 0.06, and 0.28 ± 0.06, respectfully and were within the range of previously reported results (Boldt et al., 2018; Torres-Vázquez et al., 2018).

Genetic Correlations

Genetic correlations between PAP and feedlot performance traits were found to be low to moderately correlated with ADG and ADMI ranging from -0.01 ± 0.16 to 0.06 ± 0.16 and 0.28 ± 0.19 to 0.38 ± 0.19, respectfully. Consistent with these results, Maddock et al., (2010) reported in a multi-breed study, that growing beef cattle with lower PAP had decreased ADMI. Cattle who have an increased intake spend more time visiting the feed bunks consuming more feed rather than resting, causing an increased workload of their cardiovascular system (Munro et al., 2019). Pauling, (2017) reported in a study of Angus cattle that high PAP may be slightly associated with increasing growth and muscle mass. Also, a study following a group of Angus steers from a high elevation operation to a moderate elevation feedlot, concluded that while in the feedlot, fatter and larger framed cattle appeared to have high higher PAP (Neary et al., 2015).

Genetic correlations between PAP and weaning weight direct (WWD) and weaning weight maternal (WWM) were low but positive, with an average of 0.18 ± 0.10 and 0.10 ± 0.10, respectfully. Results for WWD are slightly lower than other reported values on similar cattle with estimates of 0.14 ± 0.15 to 0.20 ± 0.04 (Crawford et al., 2016; Pauling, 2017). Genetic relationship between PAP with REA, MARB, BF, HCW, and CYG was -0.30 ± 0.12, 0.01 ± 0.13, -0.07 ± 0.13, 0.15 ± 0.10, and 0.29 ± 0.13 respectfully. Pauling, (2017), described similar results from a study analyzing the genetic relationship between PAP with carcass ultrasound measurements of REA, IMF, and BF to be 0.24 ± 0.12, -0.04 ± 0.10, and -0.03 ± 0.12, respectfully. These results suggest that selection against high PAP animals will not drive disadvantageous influence on carcass traits.

Table 4.13: Estimates of residual variance (diagonal) and covariance (lower diagonal) from the analysis of pulmonary arterial pressure, average daily gain, average dry matter intake, ribeye area, and weaning weight

	PAP	ADG	ADMI	REA	WW
PAP	60.11	-	-	-	-
ADG	-0.42	0.17	-	-	-
ADMI	-3.31	0.38	9.03	-	-
REA	-0.33	0.03	0.17	0.99	-
WW	-2.24	1.83	26.46	9.90	1539.00

PAP = Pulmonary arterial pressure, ADG = Average daily gain, ADMI = Average dry matter intake, REA = Ribeye area, WW = Weaning weight

Table 4.14: Estimates of residual variance (diagonal) and covariance (lower diagonal) from the analysis of pulmonary arterial pressure, average daily gain, average dry matter intake, marbling score, and weaning weight

	PAP	ADG	ADMI	MARB	WW
PAP	60.33	-	-	-	-
ADG	-0.49	0.17	-	-	-
ADMI	-3.52	0.40	9.12	-	-
MARB	-13.98	-0.01	15.06	7539.00	-
WW	-1.58	1.77	26.37	-16.74	1543.00

PAP = Pulmonary arterial pressure, ADG = Average daily gain, ADMI = Average dry matter intake, MARB = Marbling score, WW = Weaning weight

Table 4.15: Estimates of residual variance (diagonal) and covariance (lower diagonal) from the analysis of pulmonary arterial pressure, average daily gain, average dry matter intake, back fat, and weaning weight

	PAP	ADG	ADMI	BF	WW
PAP	60.39	-	-	-	-
ADG	-0.46	0.18	-	-	-
ADMI	-3.28	0.39	8.95	-	-
BF	-0.09	0.00	0.04	0.01	-
WW	-1.84	1.67	26.14	0.50	1541.00

PAP = Pulmonary arterial pressure, ADG = Average daily gain, ADMI = Average dry matter intake, BF = Back fat, WW = Weaning weight

Table 4.16: Estimates of residual variance (diagonal) and covariance (lower diagonal) from the analysis of pulmonary arterial pressure, average daily gain, average dry matter intake, hot carcass weight, and weaning weight

	PAP	ADG	ADMI	HCW	WW
PAP	60.39	-	-	-	-
ADG	-0.36	0.17	-	-	-
ADMI	-3.01	0.34	8.88	-	-
HCW	-76.84	7.14	48.41	3442.00	-
WW	-3.59	1.60	26.60	1089.00	1544.00

PAP = Pulmonary arterial pressure, ADG = Average daily gain, ADMI = Average dry matter intake, HCW = Hot carcass weight, WW = Weaning weight

Table 4.17: Estimates of residual variance (diagonal) and covariance (lower diagonal) from the analysis of pulmonary arterial pressure, average daily gain, average dry matter intake, calculated yield grade, and weaning weight

	PAP	ADG	ADMI	CYG	WW
PAP	60.39	-	-	-	-
ADG	-0.36	0.17	-	-	-
ADMI	-3.01	0.34	8.88	-	-
CYG	-76.84	7.14	48.41	3442.00	-
WW	-3.59	1.60	26.60	1089.00	1544.00

PAP = Pulmonary arterial pressure, ADG = Average daily gain, ADMI = Average dry matter intake, CYG = Calculated yield grade, WW = Weaning weight

Table 4.18: Estimates of genetic variance (diagonal) and covariance (lower diagonal) from the analysis of pulmonary arterial pressure, average daily gain, average dry matter intake, ribeye area, and weaning weight

	PAP	ADG	ADMI	REA	WWD	WWM
PAP	25.10	-	-	-	-	-
ADG	-0.02	0.12	-	-	-	-
ADMI	2.69	0.34	3.60	-	-	-
REA	-0.92	0.13	0.38	0.39	-	-
WWD	17.86	5.46	14.68	3.13	428.70	-
WWM	9.21	1.11	16.86	1.69	-74.26	384.40

PAP = Pulmonary arterial pressure, ADG = Average daily gain, ADMI = Average dry matter intake, REA = Ribeye area, WWD = Weaning weight direct, WWM = Weaning weight maternal

Table 4.19: Estimates of genetic variance (diagonal) and covariance (lower diagonal) from the analysis of pulmonary arterial pressure, average daily gain, average dry matter intake, marbling score, and weaning weight

	PAP	ADG	ADMI	MARB	WWD	WWM
PAP	24.78	-	-	-	-	-
ADG	0.07	0.12	-	-	-	-
ADMI	2.94	0.30	3.49	-	-	-
MARB	1.00	5.73	3.59	2837.00	-	-
WWD	17.37	5.39	14.18	135.00	419.10	-
WWM	11.14	0.64	17.17	-105.60	-75.36	383.40

PAP = Pulmonary arterial pressure, ADG = Average daily gain, ADMI = Average dry matter intake, MARB = Marbling score, WWD = Weaning weight direct, WWM = Weaning weight maternal

Table 4.20: Estimates of genetic variance (diagonal) and covariance (lower diagonal) from the analysis of pulmonary arterial pressure, average daily gain, average dry matter intake, back fat, and weaning weight

	PAP	ADG	ADMI	BF	WWD	WWM
PAP	24.69	-	-	-	-	-
ADG	0.05	0.11	-	-	-	-
ADMI	3.05	0.31	3.66	-	-	-
BF	-0.02	0.01	0.05	0.00	-	-
WWD	17.45	5.39	13.14	0.03	423.90	-
WWM	9.99	1.08	21.23	0.41	-75.62	392.50

PAP = Pulmonary arterial pressure, ADG = Average daily gain, ADMI = Average dry matter intake, BF = Back fat, WWD = Weaning weight direct, WWM = Weaning weight maternal

Table 4.21: Estimates of genetic variance (diagonal) and covariance (lower diagonal) from the analysis of pulmonary arterial pressure, average daily gain, average dry matter intake, hot carcass weight, and weaning weight

	PAP	ADG	ADMI	HCW	WWD	WWM
PAP	24.71	-	-	-	-	-
ADG	0.09	0.15	-	-	-	-
ADMI	3.25	0.51	4.47	-	-	-
HCW	39.42	18.94	81.57	2642.00	-	-
WWD	21.26	5.47	14.47	555.10	431.40	-
WWM	8.29	3.66	30.91	576.90	-39.96	401.00

PAP = Pulmonary arterial pressure, ADG = Average daily gain, ADMI = Average dry matter intake, HCW = Hot carcass weight, WWD = Weaning weight direct, WWM = Weaning weight maternal

Table 4.22: Estimates of genetic variance (diagonal) and covariance (lower diagonal) from the analysis of pulmonary arterial pressure, average daily gain, average dry matter intake, calculated yield grade, and weaning weight

	PAP	ADG	ADMI	CYG	WWD	WWM
PAP	25.08	-	-	-	-	-
ADG	0.10	0.11	-	-	-	-
ADMI	3.63	0.32	3.60	-	-	-
CYG	0.42	0.05	0.27	0.08	-	-
WWD	18.56	5.51	13.39	1.32	422.60	-
WWM	10.97	1.26	25.22	2.85	-71.13	394.10

PAP = Pulmonary arterial pressure, ADG = Average daily gain, ADMI = Average dry matter intake, CYG = Calculated yield grade, WWD = Weaning weight direct, WWM = Weaning weight maternal

Table 4.23: Heritability estimates (diagonal) and genetic correlations (above diagonal) followed by standard error from the analysis of pulmonary arterial pressure, average daily gain, average dry matter intake, ribeye area and weaning weight

	PAP	ADG	ADMI	REA	WWD	WWM
PAP	0.29 (0.03)	-0.01 (0.16)	0.28 (0.19)	-0.30 (0.11)	0.18 (0.10)	0.09 (0.10)
ADG	-	0.41 (0.09)	0.51 (0.19)	0.60 (0.17)	0.75 (0.14)	0.16 (0.22)
ADMI	-	-	0.28 (0.10)	0.32 (0.23)	0.37 (0.20)	0.45 (0.26)
REA	-	-	-	0.28 (0.05)	0.24 (0.14)	0.14 (0.15)
WWD	-	-	-	-	0.18 (0.03)	-0.18 (0.12)
WWM	-	-	-	-	-	0.16 (0.03)

PAP = Pulmonary arterial pressure, ADG = Average daily gain, ADMI = Average dry matter intake, REA = Rib eye area, WWD = Weaning weight direct, WWM = Weaning weight maternal

Table 4.24: Heritability estimates (diagonal) and genetic correlations (above diagonal) followed by standard error from the analysis of pulmonary arterial pressure, average daily gain, average dry matter intake, marbling score, and weaning weight

	PAP	ADG	ADMI	MARB	WWD	WWM
PAP	0.29 (0.02)	0.04 (0.17)	0.31 (0.20)	0.00 (0.13)	0.17 (0.10)	0.11 (0.10)
ADG	-	0.40 (0.10)	0.47 (0.20)	0.31 (0.21)	0.77 (0.15)	0.10 (0.22)
ADMI	-	-	0.28 (0.11)	0.03 (0.26)	0.37 (0.20)	0.47 (0.27)
MARB	-	-	-	0.27 (0.06)	0.12 (0.15)	-0.10 (0.16)
WWD	-	-	-	-	0.08 (0.02)	-0.18 (0.12)
WWM	-	-	-	-	-	0.07 (0.02)

PAP = Pulmonary arterial pressure, ADG = Average daily gain, ADMI = Average dry matter intake, MARB = Marbling score, WWD = Weaning weight direct, WWM = Weaning weight maternal

Table 4.25: Heritability estimates (diagonal) and genetic correlations (above diagonal) followed by standard error from the analysis of pulmonary arterial pressure, average daily gain, average dry matter intake, back fat, and weaning weight

	PAP	ADG	ADMI	BF	WWD	WWM
PAP	0.29 (0.03)	0.03 (0.17)	0.32 (0.19)	-0.07 (0.13)	0.17 (0.10)	0.10 (0.10)
ADG	-	0.39 (0.09)	0.48 (0.19)	0.38 (0.19)	0.78 (0.14)	0.16 (0.22)
ADMI	-	-	0.29 (0.11)	0.33 (0.22)	0.33 (0.20)	0.56 (0.25)
BF	-	-	-	0.27 (0.06)	0.02 (0.15)	0.30 (0.16)
WWD	-	-	-	-	0.18 (0.02)	-0.19 (0.12)
WWM	-	-	-	-	-	0.17 (0.02)

PAP = Pulmonary arterial pressure, ADG = Average daily gain, ADMI = Average dry matter intake, BF = Back fat, WWD = Weaning weight direct, WWM = Weaning weight maternal

Table 4.26: Heritability estimates (diagonal) and genetic correlations (above diagonal) followed by standard error from the analysis of pulmonary arterial pressure, average daily gain, average dry matter intake, hot carcass weight, and weaning weight

	PAP	ADG	ADMI	HCW	WWD	WWM
PAP	0.29 (0.03)	0.05 (0.14)	0.30 (0.16)	0.15 (0.10)	0.20 (0.09)	0.08 (0.09)
ADG	-	0.48 (0.08)	0.61 (0.14)	0.94 (0.06)	0.67 (0.13)	0.47 (0.17)
ADMI	-	-	0.33 (0.10)	0.75 (0.12)	0.32 (0.18)	0.73 (0.20)
HCW	-	-	-	0.43 (0.06)	0.51 (0.10)	0.56 (0.10)
WWD	-	-	-	-	0.09 (0.01)	-0.10 (0.11)
WWM	-	-	-	-	-	0.08 (0.01)

PAP = Pulmonary arterial pressure, ADG = Average daily gain, ADMI = Average dry matter intake, HCW = Hot carcass weight, WWD = Weaning weight direct, WWM = Weaning weight maternal

Table 4.27: Heritability estimates (diagonal) and genetic correlations (above diagonal) followed by standard error from the analysis of pulmonary arterial pressure, average daily gain, average dry matter intake, and calculated yield grade

	PAP	ADG	ADMI	CYG	WWD	WWM
PAP	0.29 (0.03)	0.06 (0.16)	0.38 (0.19)	0.29 (0.12)	0.18 (0.10)	0.11 (0.09)
ADG	-	0.39 (0.09)	0.50 (0.19)	0.57 (0.17)	0.79 (0.13)	0.18 (0.21)
ADMI	-	-	0.28 (0.10)	0.50 (0.20)	0.34 (0.20)	0.67 (0.24)
CYG	-	-	-	0.27 (0.06)	0.22 (0.14)	0.50 (0.15)
WWD	-	-	-	-	0.18 (0.03)	-0.17 (0.12)
WWM	-	-	-	-	-	0.16 (0.02)

PAP=Pulmonary arterial pressure, ADG=Average daily gain, ADMI= Average dry matter intake, CYG = Calculated yield grade, WWD = Weaning weight direct, WWM = Weaning weight maternal

CONCLUSIONS

High elevation cattle with high PAP could suffer from poor cardiopulmonary health, feed efficiency, and carcass quality at moderate elevation feedlots. Genetic correlation of PAP with CYG, REA, BF, and HCW suggested that cattle with lower PAP could result in a heavy muscled, leaner carcass when compared to high PAP cattle. Additionally, the more substantial genetic correlation between PAP and ADMI suggested that higher PAP cattle are less efficient at converting feed. High PAP cattle entering the feedlot at moderate elevations could be using excess energy to their pulmonary cardiovascular system resulting in a less feed efficient animal with marginal carcass quality.

IMPLICATIONS

Results from this study suggested that selection against high PAP will not negatively influence feedlot performance and carcass quality for subsequent generations. Unfortunately, cattle culled from high elevation herds due to high PAP could have a reduction in feed efficiency compared to their contemporaries when relocated to a moderate elevation feedlot for finishing. With the rising cost of finishing cattle, these findings advocate that feedlots can purchase cattle from producers that placed selection pressure on PAP in their breeding objective without subsequently having undesirable feedlot and carcass traits.

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