# The Effect of Cattle Disease on Carcass Traits

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## Introduction

Pricing cattle on the basis of carcass merit has caused the veterinary profession to re-evaluate the cost of bovine respiratory disease (BRD) and other diseases of feedlot cattle. The cost of disease when cattle are sold on a live-weight basis is confined to death loss, treatment cost, and reduced live weight. When cattle are sold on a carcass merit basis, disease has the potential to affect not only carcass weight, but also the amount, location, and ratio of muscle, fat, and water.

Undifferentiated BRD is the primary cause of feedlot cattle morbidity and mortality in the first 45 days after arrival at a feeding facility (Vogel and Parrott, 1994; Edwards, 1996). Edwards (1996) reported that 65 to 80% of morbidity within a feeding period occurred in the first 45 days and 67 to 82% of the total morbidity was due to respiratory disease. Mortality percentage ranged from 0.57 to 1.07% of all cattle received and respiratory disease accounted for 46 to 67% of deaths (Edwards, 1996). Vogel and Parrott (1994) reported that the mean monthly mortality rate due to respiratory disease for the feedlots where data was collected (Jan. 1990 to May 1993) was 0.128% (1.28 respiratory mortalities per month per 1,000 head on feed). Forty-four percent of all mortalities were due to respiratory disease. Digestive disorders, primarily acidosis, were the second highest cause of death, accounting for 25.9% of all mortalities (Vogel and Parrott, 1994).

In their review of feedlot cattle growth, Owens et al. (1995) summarized that rate and composition of tissue accretion may be controlled by chronological age, physiological age, energy intake, hormonal status, relative turnover of tissues, cell number, and cell activity. Disease could conceivably impact all of these control processes except chronological age. Mechanisms linking disease and carcass traits are not established at this time. Further research is required to understand how cattle disease affects carcass traits.

## Relationship between lung lesions present at slaughter and carcass traits

There is growing evidence that previous or active cases of respiratory disease influence carcass traits such as carcass weight, marbling, and subcutaneous fat cover (McNeill et al., 1996; Gardner et al., 1999; Stovall et al., 2000; Roeber et al., 2001). Gardner et al. (1999) found that 33% of steers in their study had lung lesions indicative of BRD. Steers with lung lesions had lighter hot carcass weight, lower dressing percent, less internal fat, and lower marbling scores ($P < 0.02$) than steers without lesions. They also tended to have less external fat and smaller longissimus muscle area ($P < 0.15$) than steers without lung lesions. Steers with lung lesions and active bronchial lymph nodes, indicative of active BRD at the time of slaughter, had lower ($P < 0.01$) dressing percentages than steers with lung lesions that had inactive bronchial lymph nodes (Gardner et al., 1999).

Carcasses from steers without respiratory tract lesions had a greater degree of marbling than carcasses from steers with lesions ($P < 0.01$; Gardner et al., 1999). Additionally, carcasses from steers with lung lesions and active bronchial lymph nodes tended to have lower marbling scores than carcasses from steers with lung lesions but without active bronchial lymph nodes ($P<0.06$; Gardner et al., 1999). As a result, steers that had lungs with active bronchial lymph nodes tended to produce a higher percentage of USDA. standard grade carcasses at the expense of USDA choice and USDA select grade carcasses ($P<0.07$; Gardner et al., 1999). Longissimus

shear force values for steaks aged 7 d were lower (P= 0.05) from steers without lung lesions than those for steaks from steers with lung lesions; however, shear force values were similar for steaks aged 14 or 21 days (Gardner et al., 1999).

**Relationship between clinical signs of BRD and carcass traits**

Gardner et al. (1999) reported that 37% of cattle that had clinical signs consistent with BRD and that were treated with appropriate antimicrobial therapy during the feedlot phase of production had lung lesions at slaughter. In comparison, 29% of cattle not identified with clinical signs of BRD had lung lesions. This apparent lack of strong association between clinical signs of BRD and lung lesions has been reported by others (Wittum et al., 1996; Buhman et al., 2000). The lack of lung lesions in cattle diagnosed by clinical signs and treated for BRD could be due to several factors, including upper respiratory tract disease or transient lower respiratory tract disease that does not result in lung pathology, full recovery from lower respiratory tract disease with complete resolution of lung lesions, or incorrect clinical assessment for the presence of BRD. In contrast, the presence of lesions at slaughter in the lungs of cattle not diagnosed with BRD could be due to respiratory tract disease that was not accompanied by clinical signs of BRD, chronic lung damage that occurred due to a BRD event prior to the time period of the investigation, or incorrect clinical assessment for the absence of BRD. Not all feedlot calves infected with known respiratory tract pathogens, as evidenced by seroconversion during finishing, are identified as having clinical signs of BRD (Martin and Bohac, 1986). It is not known if these subclinical infections could result in visible lung damage at slaughter or if they influence carcass traits.

When Gardner et al. (1999) used the presence of clinical signs consistent with BRD rather than lung lesions to indicate BRD incidence, they found that steers with clinical signs of BRD had lower hot carcass weight, less external and internal fat, and more desirable yield grades than steers without clinical signs of BRD. In contrast to steers with lung lesions, steers with clinical signs of BRD tended to have larger (P= 0.12) longissimus muscle area than untreated animals (Gardner et al., 1999). Evidence that marbling score was lower in carcasses from steers with clinical signs of BRD compared to carcasses from steers without clinical signs of BRD was present (P= 0.16), but was not as strong as evidence for the difference between carcasses from steers differentiated by the presence or absence of lung lesions (P<0.01; Gardner et al., 1999). When using clinical signs of BRD as the criterion for BRD incidence, Gardner et al. (1999) found that steers treated for BRD at the feedyard tended to have a higher percentage of carcasses that graded USDA standard and a lower percentage that graded USDA choice or USDA select than steers that were not treated, but the mean quality grade did not differ (P=0.93). There was no difference in dressing percent between steers with or without clinical signs of BRD (Gardner et al., 1999).

**Effect of prolonged or repeated treatments for BRD on carcass traits**

Some investigators have indicated that cattle treated more than one time for BRD had more pronounced negative growth and carcass effects than cattle treated only once (Gardner et al., 1999; Stovall et al., 2000; Roeber et al., 2001). Gardner et al. (1999) reported that steers treated for BRD only once gained faster (P< 0.05), had more external and internal fat (P< 0.01), had a higher dressing percentage (P< 0.06), a heavier carcass (P< 0.07), and a higher numerical yield grade (P= 0.07) than steers treated more than one time for BRD. There was no difference in longissimus muscle area or Waner-Bratzler shear force for steaks between steers treated once or

more than once for BRD (Gardner et al., 1999). Cattle treated more than once tended to have lower marbling scores than those treated only once (P=0.15).

In a study of 273 steer calves, Roeber et al. (2001) found no difference in carcass traits between cattle that had never been treated for BRD and those that had received one treatment. In contrast, they found that steers receiving two or more treatments for BRD had lower hot carcass weight, marbling score, dressing percent, and yield grade ($P< 0.05$) compared with cattle not diagnosed with BRD (Roeber et al., 2001). Warner-Brazler shear force values and palatability of longissimus steaks were not associated with whether or not the steer had been treated for BRD, or the number of BRD treatments (Roeber et al., 2001). Another study found that feedlot heifers treated more than one time for BRD had lower marbling scores than heifers never treated ($P<0.02$), which resulted in a 37.9% reduction in the percentage of carcasses grading USDA choice or above (Stovall et al., 2000).

**Signals regulating muscle growth and fat deposition:**

The regulation of muscle growth and fat deposition in beef cattle has not been fully elucidated. Hormones such as insulin, insulin-like growth factor I (IGF-I), growth hormone (GH), thyroid hormone, and leptin contribute to the partitioning of nutrients for tissue growth (Anderson et al., 1988; Rosemberg et al., 1989; Istasse et al., 1990; Trout and Schanbacher, 1990; Enright et al., 1993; Henricks et al., 1994; Rohner-Jeanrenaud 1999; Vesstergaard et al., 2003). There is also evidence that disease mediators such as interleukin-1$\alpha$, interleukin-1$\beta$, and tumor necrosis factor $\alpha$ (TNF$\alpha$) (cytokines) can also play a role in nutrient partitioning (Kelley et al., 1993;Williams et al., 1997; Raina and Jeejeebhoy, 1998; Pond, 2002; Pond and Mattacks, 2002; Broussard et al., 2003).

In healthy growing bulls, serum concentrations of GH and IGF-I are negatively correlated with carcass fat percentage, fat accretion rate, and fat thickness; moreover IGF-I concentrations were positively correlated with percentage of carcass protein (Anderson et al., 1988). Carcass weight, dressing percentage, and proportion of lean meat in the carcass of fattening bulls, is positively correlated with the total peak area or peak amplitude of growth hormone secretion (Istasse et al., 1990). In sheep, exogenous GH decreases fat content and increases the protein and water content of carcasses (Bass et al., 1991). Investigation of the mode of action of growth promoting implants has indicated that GH, IGF-I, and IGF-I binding proteins (IGFBP) play roles in mediating the growth response to both endogenous and exogenous estrogens and androgens (Gopinath and Kitts, 1984; Grigsby and Trenkle, 1986; Johnson et al., 1996; Trenkle, 1997).

In some studies, insulin concentration is positively correlated with fat content in the carcass and negatively correlated with the proportion of muscle (Istasse et al., 1990). In other studies, insulin did not appear to play a definable role in body composition (Anderson et al., 1988) or appeared to have a negative relationship with marbling (Mir et al., 2002).

Thyroid hormones ($T_3$ and $T_4$) are important mediators of metabolism throughout the body. The plasma concentrations of tri-iodothyronin ($T_3$) and tetra-iodothyronin ($T_4$) and their ratios with IGF-I were negatively correlated with carcass muscle mass (Istasse et al., 1990). Similarly, steers with higher plasma $T_4$ had increased fat deposition but tended to have lower protein deposition than steers with lower $T_4$ levels (Rumsey et al., 1987). In contrast, Enright et al. (1993) found that treatment with thyrotropin-releasing hormone (TRH) did not affect carcass weight, dressing percentage, backfat thickness, or internal organ weight; however, a combination of GH-releasing factor and TRH reduced fat score and increased longissimus muscle area relative to no hormonal treatment or treatment with either GH-releasing factor or TRH alone.

Cortisol directly inhibits pituitary thyroid stimulating hormone (TSH) and may suppress 5' deiodinase, which converts the less active $T_4$ to $T_3$ (Sharpe, 1986).

Glucocorticoids cause protein catabolism and support increased lipid storage (Rohner-Jeanrenaud, 1999). Investigations testing the effect of exogenous glucocorticoids such as dexamethasone on carcass characteristics have shown that a single injection of dexamethasone at least 33 days pre-slaughter resulted in increased marbling score in one study (Brethour, 1972). Similarly, injecting bulls with dexamethasone either once or twice within 92 days of slaughter resulted in higher mean values for marbling and quality grade than untreated controls (Dicke et al., 1974). In contrast, chronic exposure to dexamethasone via a subcutaneous implant did not enhance intramuscular fat deposition (Corah et al., 1995). Steers treated with a dexamethasone implant did however have greater external fat thickness, a higher dressing percent, and a larger longissimus muscle area compared to untreated controls (Corah et al., 1995). In trials using rats, infusion with corticosterone caused muscle catabolism (Raina and Jeejeebhoy, 1998).

The cytokine TNFα is released by activated macrophages in response to inflammation or infection (Webel et al., 1997). Very low physiological concentrations of TNFα act on muscle cells to interfere with protein synthesis and muscle cell development by inducing a state of IGF-I receptor resistance (Broussard et al., 2003). Even though IGF-I is a strong inducer of protein syntheses in myoblasts, presence of proinflammatory cytokines somehow interfere with the IGF-I promotion of muscle growth (Broussard et al., 2003). The mechanism of this interference may be that hormones and cytokines appear to share some intracellular substrates and this indicates that they might be involved in the regulation of each other (Hirosumi et al., 2002; Broussard et al., 2003). Receptors for some cytokines (interleukin-2, interleukin-4, interleukin-9, and interferon-γ) use intracellular docking molecules that were first identified for the insulin receptor (White and Yenush, 1998). Therefore, the stimulation of TNFα impairs activation of signaling elements of the insulin receptor (Hirosumi et al., 2002).

Stimulation of the pig's immune system results in increased plasma levels of cytokines and cortisol (Webel et al., 1997). These metabolically active molecules appear to be involved in the reduction of feed intake, increased muscle protein degradation, and decreased growth in diseased or immunologically challenged pigs (Webel et al., 1997).

Whether disease affects carcass traits in beef cattle by influencing the signals regulating muscle growth and fat deposition has not been directly investigated. Potential links between infectious disease, cytokines and cortisol, and body composition can be made, but much more investigation is required to test these links.

**Regulatory control of fat metabolism**

The regulatory mechanism and maturing pattern for intramuscular fat deposition may be different than for other fat depots (Belk, et al., 1993). Brethour (2000) found that the correlation between backfat thickness and marbling score was low ($r^2 = 0.07$-$0.16$). This weak relationship and differences in rate of fat accretion over time indicate that the subcutaneous fat depot and the intramuscular fat depot accumulate lipid independently and are possibly controlled by different mechanisms. Smith and Crouse (1984) found that *in vitro*, subcutaneous adipose tissue preferred acetate for fat synthesis, while muscle preferred glucose for the synthesis of intramuscular fat. They hypothesized that reduced glucose availability may decrease marbling potential (Smith and Crouse, 1984).

Fat can be mobilized as an energy source in response to a metabolic need such as response to inflammation, but different fat depots respond differently (Pond 2002; Pond and

Mattacks 2002). Adipocytes of lymph node-containing fat depots undergo lipolysis in response to inflammation of the enclosed lymph node. This response is probably mediated by cytokines (Pond, 2002). Intermuscular (seam) and intramuscular fat may also respond to similar paracrine signals of inflammation, but external fat that is not associated with lymph nodes does not appear to undergo lipolysis under the same conditions (Pond, 2002).

**Potential effects of anorexia on carcass characteristics**

*Direct Effects* - Animals infected with pathogenic microorganisms show various behavioral symptoms of sickness including lethargy, anorexia, adipsia, and reduced social interactions (Kelley et al., 1993; Dantzer, 2001). Cytokines and endotoxin induce sickness behavior in the form of reduced food intake and decreased social interactions (Dantzer, 2001). In trials using rats, infusion with TNFα caused weight loss due to anorexia (Raina and Jeejeebhoy, 1998). Interleukin-1α, interleukin-1β, and TNF-α are the most potent proinflammatory cytokines to induce behavioral alterations and it appears that different components of sickness behavior are mediated by different cytokines (Dantzer et al., 1998). Eating and drinking behavior is affected by respiratory tract disease in feedlot cattle (Sowell et al., 1999; Buhman et al., 2000). Buhman et al. (2000) found that calves that had lung lesions at slaughter had lower frequency of eating 11 to 27 days after arrival but had greater frequency of eating 28 to 57 days after arrival than calves without lung lesions. Similarly, Sowell et al. (1999) found that healthy steers had more daily feeding events than morbid steers over a 32 day receiving trial. While Buhman et al. (2000) found that sick calves had greater frequency and duration of drinking 4 to 5 days after arrival than calves that were not sick, Sowell et al. (1999) did not detect a difference in daily time spent at the water trough between healthy and sick calves.

Bulls that were restricted to an intake of 1.5% of body weight for 84 days and then realimented with an intake of 3.2% of body weight until slaughter at 422 kg (928 lbs) had less fat but the same amount of lean tissue as bulls consistently fed 3.2% of body weight (Henricks, et al., 1994). The bulls that had been restricted early in the feeding period required 35 more days on feed than non-restricted cattle to reach the same slaughter weight. During the feed restriction period, serum GH concentration was higher in restricted bulls than in unrestricted bulls on feed the same number of days. Conversely, restricted bulls had lower serum IGF-I concentration than unrestricted bulls. Once intake restriction ended, serum GH and IGF-I were similar among treatment groups. Henricks et al., (1994) point out that multiple signals appear to interact to regulate nutrient partitioning and tissue growth, and the GH:IGF-I ratio plays a central role. Although feed intake reduction has not been reported to last 84 days in sick cattle, the inversion of the GH:IGF-I ratio reported by Henricks et al (1994) may occur during the time period that sick cattle are anorexic and this may contribute to changes in carcass composition of sick cattle.

Ward et al. (1992) found that fasted cattle have higher serum cortisol concentrations than do fed cattle. Cortisol may be involved in anorexia-associated decreased carcass weight and fatness through decreased thyroid hormone activity (Sharpe, 1987), and increased protein catabolism (Rainia and Jeejeebohoy, 1998),

Brethour (2000) reported that animals starting a finishing period with little marbling usually fail to grade USDA choice after feeding periods of less than 200 days. It is possible that feedlot cattle that become sick early in the finishing period have alterations in the deposition of intramuscular fat that cause them to have similar constraints on carcass composition during finishing.

*Indirect Effects* **-** The disruption in feed intake experienced by sick cattle may be sufficient in some individuals to cause them to have fewer effective days on feed than their penmates. Sickness may have an indirect effect on carcass composition because rate and extent of fat deposition changes with days on feed.

Brethour (2000) used serial ultrasound to generate models of marbling and backfat thickness changes during the finishing period. He found that an exponential model provided the best fit for serial scans of backfat thickness over time while a power function provided the best fit for predicting marbling score. However, a consensus on the actual pattern of fat accretion in feedlot cattle has not been reached. The equation that best fit Brethour's (2000) serial ultrasound measurements depicted slow deposition of marbling early in the feeding period followed by an increased rate of intramuscular fat deposition after cattle reach low choice. In contrast, Van Koevering et al. (1995), using serial slaughter measurements, reported that marbling values reached a plateau and tended not to increase with more days on feed. Using similar techniques, Duckett et al. (1993) concluded that marbling deposition was non-linear during finishing. They reported a doubling of intramuscular fat content in the longissimus between 84 and 112 days on a high concentrate diet and then a plateau in marbling score thereafter. To describe deposition of external fat over time during the finishing phase, Camfield et al. (1997), Trenkle (1998), and Brethour (2000) reported that carcass backfat increased exponentially with time on feed, while Van Koevering et al. (1995) and May et al. (1992) reported a linear increase.

If reduced feed intake or other metabolic signals decrease the effective days on feed of sick cattle, the sick cattle should be expected to have less external and intramuscular fat compared to their penmates when the entire pen is slaughtered as a group. External fat deposition appears to proceed in an exponential or linear fashion (Camfield et al., 1997; Trenkle, 1998; Brethour, 2000). As such, reduced effective days on feed should consistently result in reduced backfat thickness of sick animals compared to health animals in the same pen. Marbling may be reduced in sick animals compared to healthy penmates if the pen is slaughtered during or shortly after the time when most of the pen has gone through the period of rapid intramuscular fat deposition (Duckett et al., 1993; Van Koevering et al., 1995). Alternatively, if the pen is slaughtered well into the period when most of the pen has reached a plateau in marbling score, the sick animals may have enough time to compensate and differences in marbling score between sick and healthy animals may be negligible.

**Potential mechanism for metabolic disease effect on carcass characteristics**

Very little information is available describing the interactions between acidosis and feedlot performance or carcass traits. One trial using lambs as a model found that a single bout of acidosis depressed rate of liquid passage and rate of absorption of volatile fatty acids (VFA) for several months (Krehbiel et al., 1995). If VFA absorption is disrupted by a single acute bout of acidosis, then growth performance and fat deposition could be affected through the finishing phase of feedlot cattle.

**Summary**

Increasing evidence indicates that BRD and possibly other diseases of feedlot cattle can have detrimental affects on carcass weight, rib eye area, marbling, and tenderness. The negative effects appear to be more severe in animals with prolonged or multiple episodes of sickness compared to animals that become sick for a short period of time and then recover. A clear mechanistic pathway linking disease to changes in carcass traits has not been made. Three

theories considered in this review are: 1) a change in metabolic signals such as cytokines and cortisol could have an effect on carcass composition through modification of hypothalamic secretions of TRH, by inhibition of IGF-I and insulin actions on muscle and fat tissues, and by direct protein catabolism and lipolysis. 2) disease-induced anorexia causing a decrease in serum IGF-I and an increase in serum GH which induces an irreversible change in the partitioning of nutrients for tissue deposition, and 3) an indirect (and reversible) effect of anorexia whereby sick cattle are on feed for fewer effective days than penmates that do not become sick. Other pathogen or immune-mediated responses to disease, as well as interactions among hormones and cytokines, may influence nutrient partitioning and body composition but have yet to be described.

The use of carcass merit to determine the value of fed cattle provides an improved economic signal of the cost of BRD and possibly other cattle diseases. The value of disease avoidance as well as rapid diagnosis and treatment of disease increases when cattle are sold on carcass merit basis because of the negative effects of disease on carcass traits.

Table 1. Potential mechanisms for disease-induced changes in carcass composition

| Disease Response | Mechanism for influencing carcass composition |
|---|---|
| ↑ Cortisol | Inhibit thyroid stimulating hormone (Sharpe, 1986)<br>Suppress 5' deiodinase (decreased conversion of T4 to T3) (Sharpe, 1986)<br>Muscle catabolism (Raina and Jeejeeboy, 1998) |
| ↑ Tumor necrosis factor α | Decreased protein synthesis (Broussard et al., 2003)<br>Insulin like growth factor I receptor resistance (Broussard et al., 2003)<br>Impaired insulin activity (Hirosumi et al., 2002)<br>Preferential lipolysis of inter- and intra-muscular fat over subcutaneous fat during inflammatory response (Pond, 2002) |
| Anorexia | Increased growth hormone (Henricks et al., 1994)<br>Decreased serum insulin like growth factor I (Henricks et al., 1994)<br>Decreased effective days on feed |

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