THE EFFECT OF RESPIRATORY DISEASE ON THE PERFORMANCE OF CATTLE IN TWO SOUTH AFRICAN FEEDLOTS

by

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Summary

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Bovine Respiratory disease (BRD) accounts for the highest proportion of morbidities and mortalities in feedlot cattle. Since both clinical and subclinical disease is known to affect growth performance, it is clear that both should be accounted for in estimating the overall effect of BRD on performance in feedlot cattle. To our knowledge there have been no studies estimating the true impact of BRD on the economy of the South African feedlot industry, including both the direct costs of treatment, labour and mortalities and the hidden costs of lower gains due to BRD.

This was an observational study, utilising predominantly routinely collected data from two commercial cattle feedlots. Assessment of the effect of treatment for BRD on outcome variables (performance parameters and prevalence of lesions) took the form of a prospective cohort study. Assessment of the effect of lung lesions at slaughter on performance took the form of a cross-sectional study, in which the presence of lung lesions and performance parameters were recorded at slaughter. Assessment of the overall effect of BRD on performance was then done using a combined case definition (treatment for BRD and/or lung lesions present at slaughter).

Slaughter data for 2036 animals were available for the final analysis. Mean average daily gain (ADG) for all animals was 1.504 kg for the period from processing to slaughter. Average days on feed (DOF) was 136 days. Peak incidence of respiratory disease in the feedlots occurred on Day 18 after arrival. A total of 22.7% of animals were treated for clinical respiratory disease. No mortalities occurred due to BRD during this period.

A total of 42.8% of animals had lung lesions present at slaughter. Of animals never treated for respiratory disease, 38.5% had lung lesions at slaughter. Of animals that had lung lesions at slaughter, 69.5% had never been treated for respiratory disease. Using the combined case definition, the estimated incidence of BRD during this study was 52%. It was found that pulling for BRD was associated with an overall decrease in ADG of 19 g for the whole period in the feedlot. The presence of lung lesions (bronchopneumonia and/or adhesions/pleuritis) at slaughter was associated with a decrease in ADG of 27 g for the whole feeding period. The occurrence of BRD (using the combined case definition) was associated with a decrease in ADG of 28 g for the period from processing to slaughter.

This translated into a hidden cost of R14.93 per animal in the feedlot. This was nearly equal to the direct variable cost/animal entering the feedlot of R15.40. The total loss due to BRD was estimated to be R30.30 per animal entering the feedlot with an estimated cost of about R40m per year to the South African feedlot industry.

1. Literature review

1.1 Aetiology and terminology

Bovine Respiratory Disease (BRD) of feedlot cattle is a broad term that describes any disease that affects the respiratory system. This could include the upper respiratory tract (URT), the lungs, or the pleura and diaphragm [39]. Also called Undifferentiated Fever (UF) [42], Shipping Fever or Fibrinous Pneumonia, the Bovine Respiratory Disease Complex (BRDC) is generally accepted to have some form of infectious involvement.

Several viruses and bacteria have been associated with bovine respiratory disease. Individually, these pathogens are usually incapable of causing the disease in healthy cattle [18]. Interactions among respiratory pathogens and compromise of the respiratory defence mechanisms are critical to the development of clinical BRD.

Viruses associated with BRD include: bovine herpes virus 1 and 3 (BHV 1 and 3), parainfluenza 3 (PI-3) virus, bovine viral diarrhoea virus (BVDV), bovine respiratory syncytial virus (BRSV), bovine adenovirus, bovine rhinovirus and bovine coronavirus.

The most important bacteria associated with the BRDC are *Mannheimia (Pasteurella) haemolytica, Pasteurella multocida, Histophilus somni, Mycoplasma* spp. and *Chlamydia* spp. [18].

1.1.1 Viruses

Bovine Herpes Virus-1, PI-3, BVDV, and BRSV are the common viruses associated with acute BRD [32,41]. Although these viruses can cause respiratory disease without significant interaction with other pathogens [16,18], pure single infections of these viruses are rarely observed in the feedlot [41]. They have been shown to potentiate each other in dual virus infection, causing more severe clinical signs and lesions than in infection with either virus alone [26]. Viral agents cause epithelial cellular damage by their local replication [18]. Lysis of

cells and the release of cellular debris act as mediators of inflammation, compromising respiratory defence mechanisms allowing bacterial pathogens access to the lower respiratory tract. Damage along the entire mucosal surface eliminates the capability for local antibody production, and provides suitable environments for bacterial replication[18]. Conversely, an aerosol of pathogenic respiratory bacteria has been shown to make cattle susceptible to respiratory viral infection[20]. Therefore, it should not be supposed that bacterial infection always necessarily follows a viral infection[18].

Bovine Herpes Virus 1

Bovine herpes virus 1 is a member of the genus Simplexvirus of the subfamily Alphaherpesvirinae of the family Herpesviridae. One group of the viruses, classified as subtype 1 (BHV1.1) or infectious bovine rhinotracheitis virus (IBRV), causes severe respiratory tract disease, conjunctivitis and abortion[18]. It is this subtype that is of concern in the feedlot and is considered to be the most important of the respiratory viruses in feedlot cattle [18].

The disease occurs mostly in animals over 6 months of age. Feedlot animals are particularly at high risk for infection because of the frequent introduction of susceptible animals into an enzootic situation[39]. There is a higher occurrence in feedlot cattle in the autumn and winter months.

The typical presentation of Infectious Bovine Rhinotracheitis (IBR) in feedlot cattle is often referred to as “red nose”[39]. Severe hyperaemia of the nasal and ocular mucosa is present. Depression, anorexia and nasal and ocular discharges are typically noted. The character of the discharge changes from serous to mucopurulent and a diphteritic membrane forms over the nasal and tracheal mucosae. A soft cough and teeth grinding can also be heard in affected cattle. Secondary bacterial infections are the primary concern in IBR and severe secondary bronchopneumonia often results. On post mortem the most striking lesion associated with IBR is an exudative tracheitis. Animals that recover from the disease remain infected for life [18,39,56].

Modified live vaccines, including intranasal products, appear to be very effective in preventing disease in newly received cattle. Protection from a modified live virus vaccine may be conferred within 48 hours [18].

Parainfluenza Virus

Parainfluenza virus is classified in the genus Paramyxovirus of the family Paramyxoviridae. Four serotypes have been described, but almost all infection and disease in livestock is caused by serotype 3 (PI-3) [39]. Primary disease attributable to PI-3 has been reported in yearling feedlot cattle [20].

It causes common respiratory infection in cattle with little or no clinical manifestation. However, in association with other viral and bacterial pathogens and stress inducing situations, it causes severe pneumonia in cattle [39]. Modified live and inactivated combination vaccines are available against the virus.

Bovine Respiratory Syncytial Virus

Bovine Respiratory Syncytial Virus (BRSV) is classified as a member of the genus Pneumovirus in the family Paramyxoviridae. This virus is considered to be a primary pathogen only in newly weaned cattle, but like PI-3, outbreaks have been reported in yearling feedlot cattle. The virus is ubiquitous in the cattle population and new infections commonly occur in the autumn and winter sometimes resulting in severe respiratory infection [20,39].

While prevalence of (BRSV) infection is high in the cattle population, the incidence of clinical disease is much lower [39]. Affected cattle typically show signs of infection within the first week of entering the feedlot, but infections can manifest in cattle over 30 days in the feedlot. The clinical signs include profuse lachrymation, nasal discharge and polypnoea. Intermandibular oedema has been reported [16]. Depressed mucociliary clearance resulting from a loss of cilia is responsible for the accumulation of fluid and tissue debris in the airways and alveoli, which provides an ideal environment for the growth of bacterial opportunists [39]. Modified live and inactivated combination vaccines are available for prevention of infection.

Bovine Viral Diarrhoea Virus

Bovine Viral Diarrhoea Virus (BVDV) is a member of the genus Pestivirus in the family Flaviviridae. It is a single stranded RNA virus that develops transcription errors that do not preclude virus replication, and thus has a high mutation rate [39]. Depending on the effect on tissue cultures there are two biotypes designated as non-cytopathic and cytopathic. The non-cytopathic type is most common [39]. This type can cross the placenta and invade and establish persistent infection in the foetus, which is crucial for spread of the virus. Cytopathic phenotypes are fairly rare and are believed to arise from mutations of non-cytopathic phenotypes. Mucosal disease is caused by superinfection with a cytopathic biotype of animals already persistently infected with noncytopathic biotype. There is no correlation between phenotype and virulence of the isolated strains, in fact the most pathogenic strains of BVDV are noncytopathic [39]. Type I and Type II genotypes are distinguishable on the basis of antigenic and genetic differences [39].

Clinically, BVDV can manifest as a mild disease with fever, inappetence, and mild diarrhoea followed by a rapid recovery in a few days. The mucosal form of the disease is characterised by the sudden onset of clinical disease in animals from 6 to 24 months of age which were infected early in foetal life. Affected animals are depressed and anorexic and salivates profusely. A profuse watery diarrhoea occurs and the faeces contain mucous and blood. Erosions can occur inside the lips, on the gums, on the dental pad, hard palate and on the tongue. Mortality rate is usually 100%.

The prevalence of infection is high, but the incidence of clinical mucosal disease is low. Of cattle over one year of age, 60 – 80% have serum neutralizing antibodies to the virus [39]. Young cattle which are persistently infected with a noncytopathic strain of the virus are the major source of infection in a herd. The mean prevalence of persistently infected animals in herds is about 1-2% [39]. The prevalence of persistently infected animals amongst calves arriving at a large feedlot in South Africa was recently found to be about 0.6%. (Thompson P N, Henson A, Schultheiss W A, unpublished data)

BVDV can be transmitted directly between animals or indirectly via flies, fomites etc [39].

Immunosuppression plays an important role in the pathogenesis of BVDV. As a result the virus has the potential to enhance disease by other pathogens or to precipitate illness by opportunistic pathogens. By depleting the host's leucocytes and suppressing leucocyte function, BVDV may enhance replication and distribution of other infectious agents, resulting in a severe respiratory disease [18,39].

Modified live and inactivated vaccines are available for the prevention of BVDV infection.

Bovine Adenovirus

Bovine adenoviruses (BAV) are members of the genus Mastadenovirus of the family Adenoviridae. All BAV's are probably transmitted by direct contact or by aerosol. Shedding takes place by lachrymal and nasal secretions as well as in faeces. Some strains have been isolated from faeces and thus may be important in intensive production units. Clinical signs may include respiratory signs together with colic and diarrhoea. Vaccines against BAV are not commonly used for feedlot cattle [18,39].

Bovine Rhinoviruses

The Rhinovirus genus belongs to the family Picornaviridae. Rhinoviruses do not cause economically important diseases in livestock, but it may be involved in cases of respiratory disease where other contributing factors are present [18]. Control by vaccination has so far been deemed unnecessary [18].

Bovine Coronaviruses

Bovine coronavirus (BCV) belongs to the genus Coronavirus of the family Coronaviridae. It was first recognized as a cause of potentially fatal diarrhoea in calves in 1972 [28]and has been isolated from cattle involved in outbreaks of respiratory disease in North America [47]. This finding has prompted concern about the possible role BCV may play in the bovine respiratory disease complex of feedlot cattle [47].

Coronaviruses are endemic and most cattle are infected [39]. Although there is evidence of the presence of BCV in feedlot cattle, it is still unclear what role it may play in BRDC because the virus also can also be isolated from apparently healthy cattle [28]. It has been

shown that cattle shedding the virus and seroconverting in the initial 28 days after arrival are at increased risk of developing respiratory disease, compared to cattle not shedding the virus or seroconverting [28]. No vaccines are commonly used in feedlots at this stage.

1.1.2 Bacteria

Bacteria and bacteria-like organisms commonly associated with bovine respiratory disease are *Mannheimia haemolytica*, *Pasteurella multocida*, *Histophilus somni*, *Mycoplasma* spp. and *Chlamydia* spp. [18]. Other bacteria that have less frequently been isolated from pneumonic lungs include *Salmonella* spp., *Streptococcus* spp., *Staphylococcus aureus*, *Escherichia coli*, *Neisseria* spp. and *Listeria monocytogenes* [56]. In general bacteria are not regarded as primary pathogens causing BRD in healthy, unstressed cattle [56].

Mannheimia haemolytica and *Pasteurella multocida*

Pneumonic pasteurellosis is a term used specifically for pneumonia caused by *Mannheimia haemolytica* (formerly *Pasteurella haemolytica*) and/or *Pasteurella multocida*. *Mannheimia haemolytica* serotype 1 is by far the most common organism recovered alone or in combination with other organisms in cases of bovine respiratory disease [12,18,32,56]. In South Africa however, isolation of *P. multocida* is becoming more frequent and in 2003 it was the most common bacteria isolated in cases of respiratory disease outbreaks in feedlots (Maryke Henton, personal communication). *Pasteurella multocida* may be more important in respiratory disease of younger cattle [56]. Both organisms inhabit the tonsils and nasal passages of healthy cattle as part of the normal bacterial flora [13,14,15]. After transport or during viral-induced illnesses, *M. haemolytica* serotype 1 can undergo rapid selective growth in the nasopharynx. This marked population increase is a likely prerequisite for the onset of pneumonic pasteurellosis [10,11,15].

Histophilus (Haemophilus) somni

Haemophilosis together with pasteurellosis are the two most important causes of morbidity and mortality in large feedlots in western Canada [50,51]. *Histophilus somni* has been reported more commonly in fatal cases of BRD in the colder climates of North America [18]. There is

some controversy about its role in BRD in moderate climates [20]. In South Africa, *H. somnis* infections constitute about one third of bacterial isolates from respiratory disease outbreaks in feedlots (Maryke Henton, personal communication). *Histophilus somni* myocarditis is an increasingly important cause of death in feedlot cattle in North America [55]. *Histophilus somni* can be isolated as a normal commensal from the mucous membranes of the respiratory and reproductive tracts of cattle [55].

Mycoplasma bovis

Mycoplasma bovis infections may present as multiple sites of arthritis following a respiratory outbreak and as isolation of *M. bovis* from respiratory mortalities. *Mycoplasma bovis* may have an immunosuppressive effect. Experimental inoculation of *M. bovis* has resulted in suppression of both cell-mediated and humoral immune responses [3]. *Mycoplasma* spp. are not considered primary pathogens of yearling cattle [3]. This organism is frequently isolated in association with other bacterial pathogens and may increase the chronicity of a lung lesion.

1.2 Epidemiology and predisposing factors

Bovine respiratory disease accounts for the highest proportion of morbidities and mortalities in feedlot cattle in North America [2,6,34,38]. Of all cattle treated, respiratory disease accounts for up to 83% of the total morbidity, although this figure is most commonly in the 15 – 45% range [41,46]. Bovine Respiratory Disease can account for up to 55% of mortality in North American feedlots [17,41,46,53].

All cattle entering the feedlot are at risk of BRD. On arrival, animals from various sources are co-mingled, exposing susceptible cattle to infectious respiratory pathogens. In addition to this, their immune systems are greatly compromised due to stressors (see below) [59]. Peak incidence of respiratory disease in feedlots occurs shortly after arrival in the feedlot, commonly within the first 35 days [2,9,59]. The highest BRD incidence occurs in the autumn and winter months [41].

A stressor can be defined as any stimulus, internal or external, chemical, physical or emotional, that excites neurones of the hypothalamus to release corticotrophin hormone at rates greater than would occur at that time of the day in the absence of the stimulus [39]. Excessive corticotrophin release leads to an increase in the synthesis of cortisol and this may lead to the suppression of the immune system [39]. Chronic stress disrupts physiological homeostasis including impaired cellular and humoral immune function and can cause digestive and respiratory dysfunction [41].

Weaning, transportation, dehydration, starvation, fatigue, processing, temperature fluctuations and co-mingling all are stressors on arrival at the feedlot. One study has shown that transportation of calves 4-6 months of age, for only 4 hours, resulted in a leucocytosis with neutrophilia, a decrease in T-lymphocyte population, a suppression of lymphocyte blastogenesis and enhancement of neutrophil activity [37].

Feedlot cattle are typically required to make a dietary transition from pasture to the high energy ration offered in the feedlot. This abrupt dietary change may increase rumen production of 3-methylindole (MI), which could result in pulmonary damage [4]. It has been suggested that the small amounts of 3-MI that are constantly produced in the rumen of cattle, although insufficient to cause clinical BRD, may be sufficient to cause subclinical lung damage [4]. It is possible that low concentrations of 3-MI act synergistically with common feedlot pathogens to cause a proportion of the morbidity attributed to undifferentiated BRD in feedlot cattle [4]. In addition systemic acidosis due to rumen acidosis is thought to lead to malfunction of the pulmonary macrophages and subsequent development of BRD although this has not been proven [59].

Extreme temperatures can suppress the immune system and lead to poorer performance in cattle [39]. Very low temperatures decrease the respiratory rate and increase tidal volume, increasing pulmonary deposition of *M. haemolytica* [8]. Very high temperatures may result in increased pulmonary airflow, thus potentially increasing the risk from airborne pathogens [52]. Abrupt changes in temperature and humidity have been shown to result in increased proliferation of *M. haemolytica* type 1 after intranasal challenge [25]. This may partly explain the increased incidence of pneumonia in autumn and winter when temperature fluctuations between night and day are greatest.

Air pollution and dust particles 2-3.3 mm in diameter, can reach the alveoli where they can saturate the phagocytic capabilities of the alveolar macrophages. The pollutants in the air may potentially be deposited in the airway and reduce mucociliary clearance [8]. Noxious gases like ammonia, carbon monoxide and diesel fumes may interfere with mucociliary clearance and alveolar macrophage function [27].

It appears that cattle are more predisposed to respiratory disease compared to other species. This can be due to a variety of morphologic and physiologic factors [54]:

(1) Cattle have a reduced respiratory capacity, necessitating an increased tidal volume and rate of pulmonary airflow, which increases exposure of the alveoli to infectious, toxic or noxious agents.

(2) The total pulmonary mass is reduced, possibly enhancing susceptibility to vascular disturbances.

(3) Bovine pulmonary capillaries have high numbers of intravascular macrophages that may contribute to vascular reactivity, and the pulmonary vein has an unusually thick muscular tunic with a greater potential for venospasm and oedema.

(4) High numbers of mast cells are present in the interstitium, which may increase vascular and airway reactivity.

(5) Cattle have a poor collateral ventilation and therefore greater potential for alveolar hypoxia .

(6) The presence in cattle of the accessory (tracheal) bronchus, which arises from the lowest point of the trachea and supplies the right cranial lobe, may be important in the pathogenesis of respiratory disease due to gravitational drainage of infected secretions into the cranioventral parts of the lungs [23].

Management of feedlot cattle can play a significant role in the health status of feedlot cattle [59]. The method of feeding, time of vaccination and co-mingling of cattle from several sources were major factors contributing to health status of feeder calves [34]. Management practices in South African feedlots can differ from those in Northern America. These differences may include younger and lighter calves being brought into the feedlot and generally shorter standing times in the feedlot till market readiness. A reason for the latter is leaner meat being more in demand in local markets.

1.3 Pathogenesis of BRD

Predisposing factors may pave the way for various agents to invade the respiratory system of cattle. Bacteria, viruses, mycoplasmas and various other stimuli can evoke an inflammatory response. Although the inflammatory response is part of the natural defence of the host, tissue damage may follow the inflammatory response.

Inflammation of the lungs begins with cell injury that leads to the haemodynamic and permeability adjustments that follow. Affected vessels readily exude fluid, plasma proteins and white blood cells. In addition the products of injured tissue can themselves serve as an inflammatory stimulus [43]. Slauson *et al.* [43] report that in calves where neutrophil depletion is accomplished in experimentally induced pneumonic pasteurellosis, the calves are partially protected against the development of severe pneumonia and resultant hypoxia. These changes are therefore, in large part, neutrophil mediated. The problem is that an intense inflammatory reaction occurs in the lung in which the tissue-damaging contributions of the inflammatory host defence mechanisms are worse than the tissue-damaging contributions of the original infectious agents. Certain organisms like *M. haemolytica* may also produce their own virulence factors (of which the most important are leukotoxin and lipopolysaccharide (LPS)), which contribute to the pathogenesis of pneumonia. Alveolar macrophages are activated by LPS and destroyed by leukotoxin. An influx of neutrophils follows, with activation and destruction by leukotoxin. The release of neutrophil lysosomal products amplifies the inflammatory cascade and is responsible for the severe damage to the lung tissue that is characteristic of the disease [31]. Leukotoxin-induced platelet damage also occurs, with the release of fibrinogen and vasoactive compounds promoting thrombosis and the accumulation of fibrinogen in interstitial sites [10].

1.4 Clinical signs and diagnosis of bovine respiratory disease in the feedlot

A typical case of feedlot respiratory disease shows a fever of 40 - 41°C or more, depression, bilateral mucopurulent nasal discharge, gaunt abdomen with rumen atony,

coughing, varying degrees of polypnoea and dyspnoea and evidence of bronchopneumonia on auscultation[39]. In the early stages there are loud breath sounds over the cranial and ventral parts of the lungs. As the disease progresses these sounds become louder and more widespread over the lung field. Pleuritic friction rubs may be audible indicating adherent pleuritis. After a few days duration the dyspnoea may become worse, commonly with an expiratory grunt. A mild diarrhoea may be present in some cases. If treated early, affected cattle recover within 24 – 48 hours, but severe cases may die in spite of treatment. Some cattle may recover spontaneously without treatment. Clinical signs specific to aetiological agents were discussed under section 1.1.

Not all cattle with respiratory disease have overt clinical signs of disease. Serologic examinations of feedlot cattle have showed that subclinical infection with bacterial and viral respiratory pathogens is common[32,33].

In the typical feedlot several thousand animals per day have to be checked. Many feedlots do this twice daily. Generally there are personnel with the specific task of identifying and pulling sick animals from their home pen to go to the treatment areas. To identify an animal as being ill in a pen full of other animals requires skill and experience. Pen checkers generally first get a general overview of the animals in the pen by observing from a distance. An animal that stands far away from the feed bunk during feeding periods immediately draws attention. Animals with signs of depression, lack of rumen fill, excessive lachrymation and/or nasal discharge, rapid breathing compared to the group and coughing, will be pulled from the pen and taken to the treatment area.[41]

At the treatment area closer examination will take place. A rectal temperature of over 40°C is generally regarded as a fever. After walking to the treatment area polypnoea or even dyspnoea may be present and coughing may be more apparent. Generally thoracic auscultation is not usually performed. This could be a reason for false or misdiagnosis of respiratory disease. A diagnosis of respiratory disease is made if one or more signs specific to the respiratory system are present, with an absence of clinical signs attributable to other systems [18,32,36,40].

1.5 Pulmonary lesions

Necropsy findings in an animal that died of pneumonia will typically show marked pulmonary consolidation usually involving at least the cranioventral lung lobes [39]. The stage of pneumonia varies within the affected tissue, ranging from congestion and oedema to airway consolidation and serofibrinous exudation in the interlobular spaces. Bronchitis, bronchiolitis and fibrinous pleuritis are usually present and may be accompanied by fibrinous pericarditis. The lung is firm and the cut surface reveals haemorrhage, necrosis and consolidation. In chronic cases there are residual lesions of bronchopneumonia with overlying pleural adhesions [39,43].

Wittum *et al.* [60] found that lung damage resulting from clinical or subclinical BRD may leave persistent lesions in bovine lungs. They suggested that the examination of lung lesions at slaughter, together with treatment records, should provide reasonable estimates of the proportion of cattle that have experienced respiratory tract disease. They found that lesions at slaughter can reflect the occurrence of disease which was significant enough to decrease production, independent of previous clinically diagnosed disease. In their study, 70% of steers never treated for respiratory signs had pulmonary lesions at slaughter.

In a study conducted by Bryant *et al.* [5] lung lesions were grouped into categories: lesions that were sequels to cranioventral bronchopneumonia (CVBP), other lesions and no lesion. Collapse/consolidation, adhesions, missing lobe, abscesses, parenchymal fibrosis and emphysema were all grouped into the CVBP category if they occurred in the cranioventral lobes.

Similarly, Gardner *et al.* [17] found pulmonary lesions in 37% of animals never treated for respiratory disease. They suggested that the high incidence of lesions among animals never diagnosed with clinical BRD indicated that either lung damage occurred during a subclinical event, BRD occurred before intake into the feedlot or BRD resulted from a viral rather than a bacterial infection.

1.6 Effect of respiratory disease on performance and economic implications

Tremendous costs are incurred in the prevention and control of losses due to feedlot respiratory disease. Respiratory disease has been estimated to cost the beef industry in North America $250 - $750 million annually [41]. However no estimates of the cost of BRD in South Africa have been made. The most obvious economic losses due to BRD are prevention (vaccines and chemoprophylaxis) and treatment (medicine and labour) costs, and mortalities. Medicine costs can account for up to 8% of total production costs [19]. In the Texas A&M Ranch to Rail Summary Reports, medical costs for calves becoming sick ranged from $20.76 to $37.90 per head for the period 1992 to 2000 [44]. Repull or retreatment rates greatly impact this cost. Economic losses due to death of the animal can also be significant. The cost of mortality exceeds the cost of the calf because of processing charges, treatment costs, feed consumed and interest [44].

A less obvious loss associated with feedlot respiratory disease is reduced rate of weight gain, translating into additional days on feed required to reach market readiness. There are numerous reports of the effect of respiratory disease on subsequent performance. McNeill *et al.* [35] reported that "healthy" steers had higher gains (1.33 vs. 1.26 kg/day) and 12% more U.S. Choice carcasses than cattle identified as "sick" at some point during the finishing period. Martin *et al.* [32], Bateman *et al.* [2] and Morck *et al.* [36] reported that gains were lower for feedlot cattle treated for BRD than those that were not treated. In a 28 day study conducted by Van Donkersgoed *et al.* [51] calves that were never sick gained 1.24 kg/day, while those treated once for BRD gained 1.18 kg/day and those treated with two or more courses of therapy gained 0.69 kg/day. Gardner *et al.* [17] found that steers never treated for BRD gained 0.04 and 0.16 kg/per day more than steers treated once and more than once respectively. Bateman *et al.* [2] found that calves which had been treated for BRD gained 0.06 kg/day less than those not treated.

However, in some other studies, that based health status on clinical evaluation alone, respiratory morbidity failed to depress daily gain during the finishing phase [21,49].

As suggested by Gardner *et al.* [17] the differences between studies regarding the effect of morbidity on feedlot performance may be partly due to differences in the definition of respiratory disease (e.g. rectal temperature vs. clinical signs), whether the infection was caused by viruses or bacteria and the accuracy of clinical appraisal to detect BRD in cattle (sensitivity and specificity of diagnosis).

Unfortunately, clinical signs of disease may often go undetected in feedlot cattle. The Strategic Alliance Field Study estimated that for every calf pulled from its pen for treatment, there are likely to be two calves that experience subclinical illness [7]. Wittum *et al.* [60] found that although 35% of steers involved in a feeding trial were treated for BRD, 72% of the steers had lung lesions at slaughter, suggesting that a significant proportion of the population had experienced a subclinical respiratory tract infection. A study by Gardner *et al.* [17] found that 33% of steers had lung lesions at slaughter; the proportions were approximately equal between cattle that were treated and those that were not. Steers without lung lesions returned an average of $20.03/ head more than those with lung lesions and non-active lymph nodes. Bryant *et al.* [5] reported that lung lesions present at slaughter were associated with a decrease of 0.025 kg in average daily gain (ADG). Cranioventral bronchopneumonia lesions were associated with a 0.033 kg reduction in ADG, whereas the effects of other lesion types were not significant.

Bryant *et al.* [5] found that lesions resulting from bronchopneumonia in the cranial ventral lung lobes are the most useful indicators for determining the effect of respiratory disease on rate of gain. CVBP lesions were significantly associated with ADG, but the amount of parenchymal involvement was not associated with ADG. They suggested that this may be because the extent of parenchymal damage is not relevant to calf growth, or differences in fibrin contraction of damaged tissue, coupled with possible differences in the initial inflammatory response and healing rates, result in the final scar not being representative of the initial magnitude of infection. Their study also found several lesions not associated with average daily gain (ADG) and that disease extensive enough to cause lung lesions, results in production losses, regardless of whether the disease manifests as a clinical or non-clinical event.

It is clear therefore that the use of symptoms or treatment rates as indicators of the incidence of BRD in the feedlot is an insensitive means of measuring the incidence of

BRD in the field. It is likely that the true effects of BRD on weight gain are underestimated in studies not identifying subclinical disease.

The mechanisms by which BRD can cause production loss, may be related to the fact that any disease that takes place in the body is potentially a catabolic event [22]. The febrile response typically caused by BRD is known to accelerate protein and energy metabolism [22]. Loew [30] states that calculations of the protein or caloric cost of fever could be made in animal production situations, thereby providing an additional estimate of production loss due to fever. Smith [44] suggested that certain heat stress proteins are produced during fever that persist in blood and may depress subsequent performance.

Another factor that may contribute to lower performance is the fact that less time is spent feeding during illness and recovery. Sowell *et al.* [45] showed that steers treated for clinical disease spent 23% less time eating and made fewer trips to the feed bunk during a 32 day recovery period. Hospital rations are also less protein and energy dense with a higher fibre content.

Various stressors cause transient endocrine responses, altered products of energy matabolism, changes in appetite and growth rate, and possibly compromise of digestive and rumen function [29]. Disease challenge, although it may result in an increase in overall disease resistance, in some cases has been associated with reduced growth rate [44]. Williams *et al.* [57] showed that a high degree of immune stimulation depresses feed intake, apparent nitrogen digestibility, nitrogen retention, tissue growth and weight gain of pigs.

Since both clinical and subclinical disease is known to affect growth performance, it is clear that both should be accounted for in estimating the overall effect of BRD on performance in feedlot cattle. The only practical way of detecting subclinical disease is to record the presence of lung lesions at slaughter. A study aiming to quantify the economic effect of BRD should thus use a case definition that includes clinical disease and the presence of lung lesions at slaughter. This was suggested by Bryant *et al.* [5].

2. Problem statement

a) Although various studies have been done to measure the effect of respiratory disease on feedlot cattle performance in other countries, no formal studies have been carried out in South Africa.

b) Although other studies have looked separately at pneumonic lesions at slaughter and treatment records, to our knowledge no study has combined these two criteria to estimate the overall effect of bovine respiratory disease on feedlot cattle performance.

c) To our knowledge there have been no studies estimating the true impact of BRD on the economy of the local feedlot industry, including both the direct costs of treatment and labour and the hidden costs of lower gains due to BRD.

3. Objectives

a) To estimate the effect of respiratory disease on average daily gain and standing time of cattle in two South African feedlots.

b) To use a combined case definition, including treatment for BRD in the feedlot or the presence of pneumonic lesions at slaughter, to estimate the overall effect of BRD on performance.

c) To estimate the economic impact of BRD, taking into account the direct costs of treatment and labour and the hidden costs of lower gains due to BRD.

4. Materials and methods

4.1 Experimental design

This was an observational study, utilising predominantly routinely collected data from two commercial cattle feedlots.

Assessment of the effect of treatment for BRD on outcome variables (performance parameters and prevalence of lesions) took the form of a prospective cohort study. Animals self-selected into cohorts based on whether or not they were treated for BRD during their stay in the feedlot.

Assessment of the effect of lung lesions at slaughter on performance took the form of a cross-sectional study, in which the presence of lung lesions and the performance parameters were recorded at slaughter. The same study population was used for both components.

4.2 Inclusion criteria

a) Animals were bought in the normal course of business of the feedlot.
b) Diagnosis of BRD was according to the normal feedlot protocol.
c) Only animals with unique identification throughout the feeding period to the slaughter day were included.
d) Only animals with individual masses on record were included.
e) At the abattoir all lungs had to be identifiable with the carcass of origin.

4.3 Sample size determination

Sample size determination was done for a Student's *t*-test comparing average daily gain (ADG) between treated animals with lung lesions and treated animals without lung lesions.

The group of treated animals was used because fewer treated animals than untreated animals were expected. The sample size was calculated based on a minimum detectable difference in ADG of 0.1 kg over the feeding period and SD of 0.25 kg. The significance level (a) was set at 0.05 and the desired power (1- ß) at 0.9 (90%). Sample size was calculated using PASS 2000 power analysis and sample size software (NCSS, Kaysville, Utah).

Assuming that 75% of treated animals that recovered would still have lung lesions at slaughter, 352 treated animals were required, and conservatively assuming a treatment rate of 20%, a total sample size of 1760 animals was required. To ensure that adequate numbers for statistical significance were achieved, 3500 animals were initially included in the trial.

4.4 Experimental model system

4.4.1 Study sites

Two feedlots in Gauteng province, South Africa, were used for the trial. These feedlots were owned by the same feedlot operator and had the same management practices. One feedlot site (Site 1) was situated near Bapsfontein 30 km southwest of Pretoria and the other, (Site 2), near Krugersdorp northeast of Johannesburg. These feedlots had very high standards of record keeping, excellent management and convenient location. The type of animals bought and the climate were considered to be representative of the typical feedlot in South Africa.

4.4.2 Animal management and treatment

Trial animals were bought by the feedlot operator in the normal course of business at auctions or directly from farms from different districts all over South Africa. Predominantly steers, of no specific breed, between the age of 5 and 10 months at the start of the trial and with a body mass between 150 and 300 kg were used in the trial. This represented the typical South African feedlot animal. Animals brought into the feedlots

between 23 April 2003 and 16 May 2003 were included in the trial. This time period was chosen as it is generally the time of greatest BRD incidence in South African feedlots.

Calves were transported to the feedlot site by trucks. Before offloading (Day 0) the average body mass for each load of calves was recorded on a weighbridge. New animals were then put in receiver pens with fresh water and ad lib hay for 24 hours. On Day 1 the cattle were identified with an ear tag and individual number, weighed, an acaricide was topically applied (Ectoline, Bayer) and a freeze-dried vaccine of modified live strains of infectious bovine rhinotracheitis (IBR), bovine viral diarrhoea (BVD) virus, parainfluenza$_3$ (PI$_3$) virus and bovine respiratory syncytial virus (BRSV) was administered (Bovishield, Pfizer). A small amount of prestarter concentrate feed was given from Day 1 for 4 - 6 days, after which feeding of the starter ration commenced.

On Day 5 after arrival the calves were processed. During processing animals were vaccinated against botulism (Botuvax, Intervet SA), lumpy skin disease, (Onderstepoort Biological Products), anthrax (Anthravax, Intervet SA) and clostridial diseases (Siteguard MLG, Schering Plough Animal Health). A growth stimulant containing zeranol 36 mg (Ralgro, Schering Plough A.H.) was given subcutaneously behind the left ear, bulls were castrated with a burdizzo and tip dehorning was done. High-risk groups of animals (animals that arrived during inclement weather, had not been vaccinated and/or were weaned onto the truck) were given tilmicosin (Micotil 300, Elanco Animal Health) 10mg/kg subcutaneously as a preventative measure.

Each animal was weighed at processing. Each feedlot had a single electronic mass indicator that was used to weigh all the animals at the particular feedlot. These mass indicators were checked on a weekly basis using known weights. At the abattoir, another single instrument for all the animals was used to measure mass. This instrument was also checked on a weekly basis in the same manner as at the feedlots.

On Day 35 cattle were again weighed and separated from their original groups by mass:

- Oxen < 225 kg: small group
- Oxen 226 – 250 kg: medium group
- Oxen > 251 kg: large group

- Heifers < 220 kg: small group
- Heifers > 220 kg: large group.

Large and medium oxen started on the production ration for 7 days and then onto the finisher ration from Day 42. Heifers and small oxen started on a different ration until Day 56. On Day 56 all animals were weighed and another implant containing 140 mg trenbolone acetate and 28 mg ß-oestradiol (Revalor-S, Intervet SA) was administered to groups of cattle = 225 kg.

Feeding throughout the feedlot period was done three times daily at 06:30, 09:30 and 13:00. Feed bunks were checked just before feeding and the amount of food put out was calculated from the amount of food left in the bunk after the previous feeding.

Throughout the standing time each pen of cattle was checked daily at 06h00 and 15h00 by experienced pen checkers. Animals that appeared depressed, listless or showed any specific signs of disease were pulled from the pen and taken to the treatment area. There they were examined and their rectal temperatures taken. This information was recorded on a record sheet. According to the examiner's findings they were categorised as respiratory, bloat, abdominal pain, diarrhoea, vitamin B1 deficiency, blood stomach, tick borne disease (specified), foot problems, musculo-skeletal, eye problems or other disease (specified). Appropriate treatment, according to the feedlot's treatment program, was then administered.

Cattle that were pulled and diagnosed with respiratory disease were treated with oxytetracycline (Engemycin 10%, Intervet) 10 mg/kg body mass intravenously. Tylosin (Tylo 200, Phenix (Virbac)) 8 mg/kg body mass intramuscularly as well as flunixin meglumine (Finadyne, Schering Plough Animal Health) 2.2 mg/kg intramuscularly were added depending on the severity of disease. The oxytetracycline (and Tylosin if given on the day of pulling) treatment was repeated on the following day.

On the third day of treatment trimethoprim 40 mg/ sulphadiazene 200 mg (Norodine 24, Centaur Labs) 1 ml /16 kg body mass was administered. The medicine and dosages were recorded for each animal. These animals were then put into the hospital pen where ad lib hay, small amounts of concentrate and fresh water were available.

The decision whether or not to treat was made by the supervisor on the basis of the animal's habitus and temperature. An animal with a temperature >40 ºC was treated irrespective of other signs. An animal with a low temperature but other signs of illness was also treated. A pulled but not treated animal on the basis of the above criteria, was again assessed twelve hours later. Treated animals stayed in hospital for five days and were observed frequently for improvement. Animals not responding to treatment received additional treatments if necessary.

Any mortalities were necropsied and observations recorded by the examiner on a standard post mortem sheet. The post mortem results were checked against treatments received.

Cattle from Site 1 and 2 were taken to the finisher and abattoir site from about 90 days on feed. This site was 15 km from Site 1 and 100 km from Site 2. Here cattle were selected for market readiness by the feedlot owner and manager, who had no knowledge of each animal's pulling history.

4.4.3 Lung lesion evaluation at slaughter

Slaughtering of the trial group took place between 12 August and 17 October 2003 at an A Grade abattoir (throughput can be >100 animals/day, preslaughter, isolation, examination and laboratory facilities available and the highest standards of slaughtering and hygiene maintained). At the abattoir, cattle were stunned using a captive bolt pistol and bled. Six minutes after bleeding, the hot carcass mass was recorded. From this the adjusted slaughter mass was recorded by assuming that 7% of an animal's body mass is blood and that 50% of this is bled out after 4-6 minutes [58]. Each animal's organs were identified with the carcass using a bar code system.

Lung lesions were recorded using the results obtained by Bryant *et al.* [5] as a guide to identify significant lesions. Each lung was visually inspected for lesions and palpated for consolidation. Specific attention was paid to cranioventral lesions. Each set of lungs was assigned a bronchopneumonia score as follows:

0: no visual or palpable lesion occurred, or only hyperaemia of the cranioventral lung lobes without any consolidation
1: consolidation of up to 50% of the cranioventral lobe(s)
2: cosolidation of 51 – 100% of the cranioventral lobe(s)

Any lesions occurring elsewhere on the lung surface were recorded as other lesions and not distinguished with regard to type or area of involvement.

In addition, a pleuritis score was recorded for each set of lungs as follows:

0: no adhesions or pleuritis
1: adhesions or pleuritis present, but involving less than 50% of the lung/pleural surface
2: adhesions or pleuritis present, involving more than 50% of the lung/pleural surface

Each carcass was traceable to its original ear tag number by means of a bar code number. Slaughter speed was on average 30 animals per hour, which made proper inspection of the lungs possible.

4.4.4 Data collection and management

Raw data were recorded by hand on data collection sheets and from computer printouts at the feedlots and abattoir. They were then entered/ imported into a spreadsheet (Microsoft Excel ® 2000).
The raw data variables and additional variables calculated from the raw data are shown in Tables 1 and 2.

Table 1. Raw data variables collected from feedlots, with definitions

Variable	Definition
Ear tag number	Each calf was assigned an individual number
Mass on Day 1	Individual animal weight on first day after arrival
Processing mass	Individual mass at processing
Day 35 mass	Individual mass for each individual at 35 days on feed
Sex	Male/female
Origin	The farm/district of origin for each calf
Date of arrival	Arrival at the feedlot
Treatments and dosages received	Drugs, dose and route
Rectal temperature at hospital	For days one and three in hospital
Dates of and number of pulls per animal	
Slaughter date	
Hot carcass mass	Mass immediately after bleeding
Bronchopneumonia score	0: no lesions or hyperaemia only 1: 1 – 50% of the cranioventral lobe(s) showed consolidation 2: 51 – 100% of cranioventral lobe(s) showed consolidation
Adhesion/pleuritis score	0: no adhesions/pleuritis present 1: adhesions or pleuritis present, but involving less than 50% of lung/pleural surface 2: adhesions and/or pleuritis present involving more than 50% of lung/pleural surface
Carcass grade at slaughter	According to South African grading system Incorporates the age (using teeth) and fat distribution for each animal on visual appraisal

Table 2. Variables calculated from raw data variables, with definitions

Variable	Definition
ADG from processing day to Day 35	Average daily mass gain between Day 5 and Day 35
ADG from Day 35 to slaughter	Average daily mass gain between Day 35 and slaughter based on adjusted slaughter mass
ADG from processing to slaughter	Average daily mass gain between processing and slaughter based on adjusted slaughter mass
Adjusted slaughter mass	Hot carcass mass × 100/96.5
Days on feed (DOF)	Standing time in the feedlot from day of arrival to day of slaughter
Total cost of medicine used for each calf pulled for respiratory disease.	Drug list prices as in April 2003 [48]
Labour cost for each calf pulled for respiratory disease.	This was estimated for the amount of time spent by labourers to identify, pull and treat an animal per treatment course
Lung lesions present at slaughter	0: Bronchopneumonia score = 0 AND adhesions/pleuritis score = 0 1: Bronchopneumonia score >0 OR adhesion/pleuritis score >0
Occurrence of BRD	0: Not pulled and no lung lesions 1: Animal was pulled for BRD and/or had lung lesions at slaughter

4.5 Data analysis

Associations between treatment for respiratory disease and the presence of the various lesions at slaughter, as well as other associations between categorical variables, were assessed by cross-tabulation and analysed using Fisher's exact test (for 2×2 tables) or the Chi-squared test (for larger tables).

For the purposes of determining the effect of BRD on cattle performance, the outcome variables used were average daily gain (ADG) between processing and slaughter, ADG between processing and Day 35, ADG between Day 35 and slaughter, and total days on feed (DOF) from arrival to slaughter.

The predictor variables of interest were whether or not the animal was pulled for BRD, the presence of pulmonary lesions at slaughter, the severity of bronchopneumonic lesions at slaughter and the severity of adhesions at slaughter. Finally, the main predictor of interest was whether or not the animal had suffered from respiratory disease during its stay in the feedlot. An animal was assumed to have experienced respiratory disease if it had been pulled and treated for BRD, or if it showed either bronchopneumonic lesions or pleural adhesions at slaughter, or both.

Potential confounders that were considered were site (Site 1 vs. Site 2), sex (male vs. female), region of origin of the animal, mass at processing, and whether or not the animal was treated for diseases other than BRD during its stay in the feedlot. However, region of origin perfectly predicted site, i.e. all the animals from any given origin went to either Site 1 or Site 2, but never to both. Therefore in order to avoid problems with multicollinearity, origin was retained but site was not considered as an independent variable.

Univariable associations between each predictor variable or potential confounder and the outcome variables were first analysed using simple linear regression. Multiple linear regression models were then developed in order to estimate the effect of each of the five predictors on each of the four outcomes, i.e. a total of $5 \times 4 = 20$ multiple linear regression models. In each model the potential confounders (processing mass, origin, sex and other disease) were initially included and the model was then developed by backward elimination. Variables were retained in the model if they remained significant (Wald's $P \leq 0.05$) or if their removal resulted in a >10% change in the coefficient for the main predictor of interest.

Except for processing mass, all independent variables (predictors and potential confounders) were regarded as categorical variables and were recoded as (k-1) regular indicator (dummy) variables, where k is the number of categories (levels) represented by

the variable. For example, origin had 21 categories and was therefore included in the models as 20 binary (0/1) indicator variables.

Statistical analyses were performed using NCSS 2001 statistical software (NCSS, Kaysville, Utah) and a public-domain statistical calculator, EpiCalc 2000 (http://www.brixtonhealth.com/epicalc.html).

4.6 Economic analysis

Only variable costs were included in the economic analysis. Direct variable costs were medicine costs, labour costs and mortalities. Medicine costs were calculated at list drug prices for April 2003 [48]. Labour costs were estimated to be R2 per head per treatment course.

Direct variable costs (DVC) per animal entering the feedlot were calculated as follows:

DVC = (Total treatment costs + labour costs + mortality cost) ¸ total number of animals entering feedlot

where mortality cost was the sum of the purchase prices, treatment and labour costs and approximate feeding costs for all animals dying from BRD.

Indirect or hidden variable costs (IVC) were due to loss of production resulting from BRD. The meat price was taken at R13.00/kg at the time. Indirect variable costs per animal entering the feedlot were calculated as follows:

IVC = Reduction in ADG due to BRD ´ mean DOF ´ mean dressing percentage ´ meat price ´ incidence of BRD

The total cost of BRD was the sum of direct variable costs and indirect variable costs.

5. Results

5.1 General

Of 3641 animals entered into the study, slaughter data for 2036 animals were available for the final analysis. For a few animals data were not complete resulting in different totals for some analyses.

Mean average daily gain (ADG) for all animals was 1.504 kg for the period from processing to slaughter. Male animals on average gained 265 g/day more than females (males: 1.563 kg/day, females: 1.298 kg/day). There were significantly more males (77.7%) than females (22.3%) in the study. There was a difference in ADG between sites, which was probably due to the difference in proportion of sexes between the two sites (site 1: 75.5% male, site 2: 80.6% male) and the fact that the two sites received calves from different origins. Average days on feed (DOF) was 136 days. On average males were fed 4.58 days longer than females.

Detailed descriptive statistics for the performance outcomes are shown below in Tables 6 - 9.

5.2 Clinical disease and lung lesions at slaughter

The peak incidence (mode) of respiratory disease in the feedlots occurred on Day 18 after arrival (32 pulls), and the median number of days to first treatment was 19. By Day 35, 87% of all respiratory pulls had been made (Fig. 1).

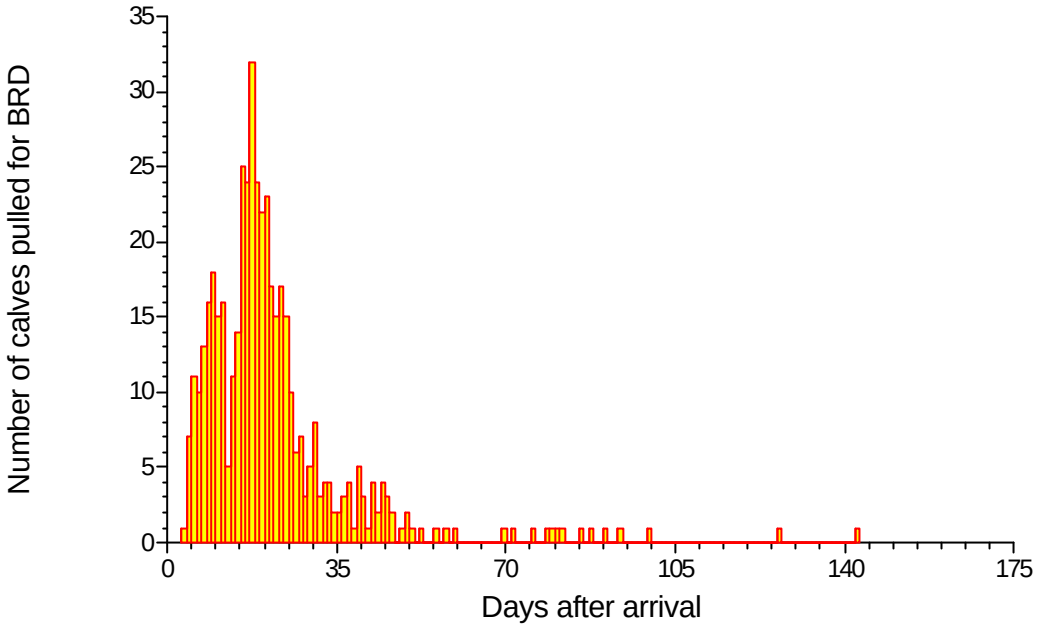

Figure 1. Numbers of calves pulled for respiratory disease on each day after arrival at the feedlot

A total of 461/2036 animals (22.6%) were pulled for clinical respiratory disease. Of these, 380 were pulled once and 81 were pulled twice or more (retreatment rate of 17.6%). A total of 870/2033 animals (42.8%) had lung lesions present at slaughter. The estimated overall incidence of respiratory disease (defined as animals diagnosed with clinical respiratory disease and/or lung lesions present at slaughter) was 1067/2033 (52.5%).

The association between the number of times an animal was pulled for BRD and the presence of lung lesions at slaughter is summarized in Table 3. Of animals never pulled for respiratory disease, 605/1571 (38.5%) had lung lesions at slaughter. Of animals that had lung lesions at slaughter, 605/870 (69.5%) had never been diagnosed with respiratory disease. Of animals pulled more than once, 54/81 (66.6%) had lung lesions at slaughter.

Table 3. The association between clinical respiratory disease and lung lesions at slaughter in feedlot cattle

Number of respiratory pulls	Any lung lesion present		Total
	No	Yes	
0	966	605	1571
1	170	211	381
> 1	27	54	81
Total	1163	870	2033

$?^2$ $P < 0.001$

The association between clinical respiratory disease and the presence of adhesions/pleuritis at slaughter is summarized in Table 4. Of animals never pulled for respiratory disease, 538/1571 (34.3%) had adhesions/pleuritis present at slaughter. Of animals with severe adhesions, 20/34 (58.8%) had never been pulled for respiratory disease. Animals pulled more than once had a 52/81 (64%) chance of having adhesions at slaughter.

Table 4. The association between clinical respiratory disease and adhesions/pleuritis at slaughter

Number of respiratory pulls	Adhesions/pleuritis score			Total
	0	**1**	**2**	
0	1033	518	20	1571
1	183	189	9	381
> 1	29	47	5	81
Total	1245	754	34	2033

$?^2$ $P < 0.001$

The association between clinical respiratory disease and bronchopneumonia score is summarized in Table 5. Of animals with severe bronchopneumonia (score of 2), 37/56 (66.1%) had never been pulled for respiratory disease. A total of 1447/1571 (92%) animals never pulled had no bronchopneumonia lesions. Only 50/462 (10.8%) of animals pulled had bronchopneumonia lesions at slaughter.

Table 5. The association between clinical respiratory disease and bronchopneumonia score

Number of respiratory pulls	Bronchopneumonia score			Total
	0	**1**	**2**	
0	1447	87	37	1571
1	340	25	16	381
> 1	72	6	3	81
Total	1859	118	56	2033

$?^2$ $P = 0.26$

5.3 Effect of BRD on performance

5.3.1 Univariable analysis

Univariable associations between predictor variables and ADG from processing to slaughter, processing to Day 35 and Day 35 to slaughter are summarized in Tables 6 – 8 respectively.

Univariable associations between predictor variables and days on feed (DOF) are summarized in Table 9. Apart from BRD, anaplasmosis was the only disease diagnosed in significant numbers during the study period.

Regardless of significance in the univariable analysis, all predictors and potential confounders were entered into the multiple regression models, with the exception of site, as explained earlier in section 4.5.

Table 6. Descriptive statistics and univariable associations of predictor variables and covariates with average daily gain (ADG) from processing to slaughter in feedlot cattle

Variable and level	n	ADG (kg)		Simple linear regression model		
		Mean	S.D.	*b*	S.E.(*b*)	*P*-value
Respiratory disease						
Yes	1053	1.490	0.250	-0.029	0.011	0.01
No	955	1.519	0.259	0 [a]	0	-
Pulled for BRD						
Yes	455	1.488	0.252	-0.020	0.014	0.1
No	1558	1.508	0.255	0 [a]	0	-
Lung lesions present						
Yes	859	1.484	0.245	-0.034	0.012	0.004
No	1149	1.518	0.261	0 [a]	0	-
Lung lesion score						
0	1837	1.506	0.255	0 [a]	0	-
1	116	1.478	0.260	-0.028	0.024	0.2 [b]
2	55	1.458	0.238	-0.048	0.035	0.2 [b]
Adhesion score						
0	1228	1.516	0.262	0 [a]	0	-
1	746	1.487	0.242	-0.029	0.012	0.02 [b]
2	34	1.401	0.223	-0.115	0.044	0.009 [b]
Processing mass						
Continuous	2013	1.504	0.255	0.001	0.0002	<0.001
Sex						
Male	1563	1.563	0.232	0.265	0.012	<0.001
Female	450	1.298	0.221	0 [a]	0	-
Anaplasmosis						
Yes	122	1.511	0.230	0.008	0.024	0.7
No	1891	1.503	0.256	0 [a]	0	-
Origin						
21 categories	2013	1.504	0.255	-	-	<0.001 [b]
Site						
Site 1	1154	1.465	0.271	-0.089	0.011	<0.001
Site 2	859	1.555	0.222	0 [a]	0	-

[a] Reference level
[b] Refers to a multiple linear regression model with only one predictor, recoded as (k-1) indicator (dummy) variables.

Table 7. Descriptive statistics and univariable associations of predictor variables and covariates with average daily gain (ADG) from processing to Day 35 in feedlot cattle

Variable and level	n	ADG (kg)		Simple linear regression model		
		Mean	S.D.	*b*	S.E.(*b*)	*P*-value
Respiratory disease						
Yes	1025	0.869	0.578	-0.156	0.026	<0.001
No	938	1.025	0.550	0 [a]	0	-
Pulled for BRD						
Yes	434	0.801	0.608	-0.183	0.031	<0.001
No	1534	0.984	0.553	0 [a]	0	-
Lung lesions present						
Yes	841	0.877	0.562	-0.117	0.026	<0.001
No	1122	0.994	0.570	0 [a]	0	-
Lung lesion score						
0	1799	0.946	0.574	0 [a]	0	-
1	110	0.856	0.533	-0.090	0.056	0.1 [b]
2	54	1.028	0.473	0.082	0.079	0.3 [b]
Adhesion score						
0	1199	0.996	0.569	0 [a]	0	-
1	730	0.867	0.563	-0.129	0.027	<0.001 [b]
2	34	0.730	0.525	-0.266	0.098	0.007 [b]
Processing mass						
Continuous	1968	0.944	0.571	-0.0002	0.0004	0.7
Sex						
Male	1530	0.998	0.560	0.244	0.030	<0.001
Female	438	0.754	0.567	0 [a]	0	-
Anaplasmosis						
Yes	121	1.061	0.481	0.125	0.054	0.02
No	1847	0.936	0.575	0 [a]	0	-
Origin						
21 categories	1968	0.944	0.571	-	-	<0.001 [b]
Site						
Site 1	1128	0.758	0.568	-0.435	0.021	<0.001
Site 2	840	1.192	0.470	0 [a]	0	-

[a] Reference level
[b] Refers to a multiple linear regression model with only one predictor, recoded as indicator (dummy) variables.

Table 8. Descriptive statistics and univariable associations of predictor variables and covariates with average daily gain (ADG) from Day 35 to slaughter in feedlot cattle

Variable and level	n	ADG (kg)		Simple linear regression model		
		Mean	S.D.	*b*	S.E.(*b*)	*P*-value
Respiratory disease						
Yes	1031	1.671	0.294	-0.003	0.013	0.8
No	941	1.674	0.295	0 [a]	0	-
Pulled for BRD						
Yes	439	1.687	0.304	0.019	0.016	0.2
No	1538	1.668	0.291	0 [a]	0	-
Lung lesions present						
Yes	844	1.660	0.285	-0.022	0.013	0.1
No	1128	1.682	0.302	0 [a]	0	-
Lung lesion score						
0	1806	1.675	0.294	0 [a]	0	-
1	111	1.671	0.301	-0.004	0.029	0.9 [b]
2	55	1.576	0.284	-0.099	0.040	0.01 [b]
Adhesion score						
0	1207	1.680	0.303	0 [a]	0	-
1	731	1.664	0.281	-0.016	0.014	0.25 [b]
2	34	1.600	0.265	-0.080	0.051	0.1 [b]
Processing mass						
Continuous	1977	1.672	0.294	0.001	0.0002	<0.001
Sex						
Male	1538	1.729	0.277	0.256	0.015	<0.001
Female	439	1.474	0.265	0 [a]	0	-
Anaplasmosis						
Yes	121	1.633	0.264	-0.042	0.028	0.1
No	1856	1.675	0.296	0 [a]	0	-
Origin						
21 categories	1977	1.672	0.294	-	-	<0.001 [b]
Site						
Site 1	1133	1.679	0.318	0.015	0.013	0.2
Site 2	844	1.664	0.259	0 [a]	0	-

[a] Reference level
[b] Refers to a multiple linear regression model with only one predictor, recoded as (k-1) indicator (dummy) variables.

Table 9. Descriptive statistics and univariable associations of predictor variables and covariates with days on feed (DOF) from processing to slaughter in feedlot cattle

Variable and level	n	DOF		Simple linear regression model		
		Mean	S.D.	*b*	S.E.(*b*)	*P*-value
Respiratory disease						
Yes	1066	140.2	18.5	6.87	0.83	<0.001
No	965	133.3	18.8	0[a]	0	-
Pulled for BRD						
Yes	461	140.8	19.7	5.11	1.00	<0.001
No	1575	135.7	18.5	0[a]	0	-
Lung lesions present						
Yes	869	140.8	17.9	6.88	0.84	<0.001
No	1162	134.0	19.2	0[a]	0	-
Lung lesion score						
0	1858	137.0	19.1	0[a]	0	-
1	117	134.8	17.9	-2.15	1.81	0.2[b]
2	56	137.9	17.6	0.90	2.57	0.7[b]
Adhesion score						
0	1243	133.8	19.0	0[a]	0	-
1	754	142.1	17.8	8.33	0.86	<0.001[b]
2	34	134.6	17.8	0.77	3.22	0.8[b]
Processing mass						
Continuous	2036	136.9	18.9	-0.07	0.01	<0.001
Sex						
Male	1581	137.9	18.6	4.58	1.00	<0.001
Female	455	133.3	19.5	0[a]	0	-
Anaplasmosis						
Yes	122	146.4	17.0	10.1	1.75	<0.001
No	1914	136.3	18.9	0[a]	0	-
Origin						
21 categories	2036	136.9	18.9	-	-	<0.001[b]
Site						
Site 1	1171	136.1	17.1	-1.89	0.85	0.03
Site 2	865	138.0	21.1	0[a]	0	-

[a] Reference level
[b] Refers to a multiple linear regression model with only one predictor, recoded as (k-1) indicator (dummy) variables.

5.3.2 Multivariable analysis

The final multiple regression models estimating the effects of the various predictors on the ADG and DOF outcomes are presented in Tables 10 – 19. For categorical predictors, estimated effects for each level are relative to the reference level as defined in each table.

Note that, for the ADG outcomes (Tables 10, 12, 14, 16 and 18) each table describes three models: ADG from processing to slaughter, ADG from processing to Day 35 and ADG from Day 35 to slaughter.

5.3.2.1 Effect of clinical respiratory disease on performance

The effect of clinical respiratory disease on ADG was significant only during the first 35 days, when it resulted in a reduction of 165 g/day (P <0.001) (Table 10). Treatment for BRD was also associated with an animal being 2.60 days longer on feed (P = 0.006) (Table 11).

Table 10. Final multiple regression models: effect of clinical respiratory disease (animal pulled for BRD) on average daily gain (ADG) in feedlot cattle

Outcome	Predictor and level	*b*	S.E.(*b*)	95% confidence interval	*P*-value
ADG from processing to slaughter	Pulled for BRD				
	Yes	-0.019	0.012	-0.043, 0.004	0.1
	No	0[a]	0	-	-
	Processing mass				
	Continuous	0.0008	0.0002	0.0005, 0.001	<0.001
	Sex				
	Male	0.265	0.013	0.239, 0.291	<0.001
	Female	0[a]	0	-	-
	Anaplasmosis				
	Yes	-0.060	0.022	-0.102, -0.018	0.006
	No	0[a]	0	-	-
	Origin				
	k=21	-	-	-	<0.001
ADG from processing to Day 35	Pulled for BRD				
	Yes	-0.165	0.027	-0.217, -0.113	<0.001
	No	0[a]	0	-	-
	Sex				
	Male	0.229	0.028	0.173, 0.284	<0.001
	Female	0[a]	0	-	-
	Origin				
	k=21	-	-	-	<0.001
ADG from Day 35 to slaughter	Pulled for BRD				
	Yes	0.024	0.015	-0.005, 0.053	0.1
	No	0[a]	0	-	-
	Processing mass				
	Continuous	0.001	0.0002	0.0006, 0.001	<0.001
	Sex				
	Male	0.260	0.016	0.228, 0.292	<0.001
	Female	0[a]	0	-	-
	Anaplasmosis				
	Yes	-0.096	0.026	-0.148, -0.045	<0.001
	No	0[a]	0	-	-
	Origin				
	k=21	-	-	-	<0.001

[a] Reference level

Table 11. Final multiple regression model: effect of clinical respiratory disease (animal pulled for BRD) on days on feed (DOF) in feedlot cattle

Predictor and level	*b*	S.E.(*b*)	95% confidence interval	*P*-value
Pulled for BRD				
Yes	2.60	0.95	0.73, 4.48	0.006
No	0[a]	0	-	-
Processing mass				
Continuous	-0.09	0.01	-0.11, -0.06	<0.001
Sex				
Male	6.00	1.06	3.93, 8.07	<0.001
Female	0[a]	0	-	-
Anaplasmosis				
Yes	4.47	1.72	1.09, 7.85	0.01
No	0[a]	0	-	-
Origin				
k=21	-	-	-	<0.001

[a] Reference level

The effect of the number of pulls on the performance outcomes is shown in Figures 2 - 5. For none of the four outcomes was there a statistically significant difference between animals treated once and animals retreated.

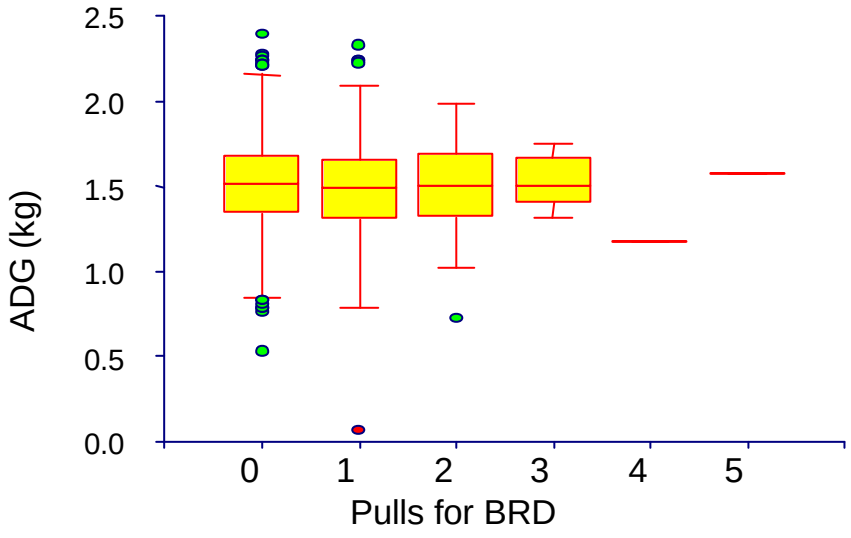

Figure 2. Effect of the number of BRD pulls on ADG from processing to slaughter

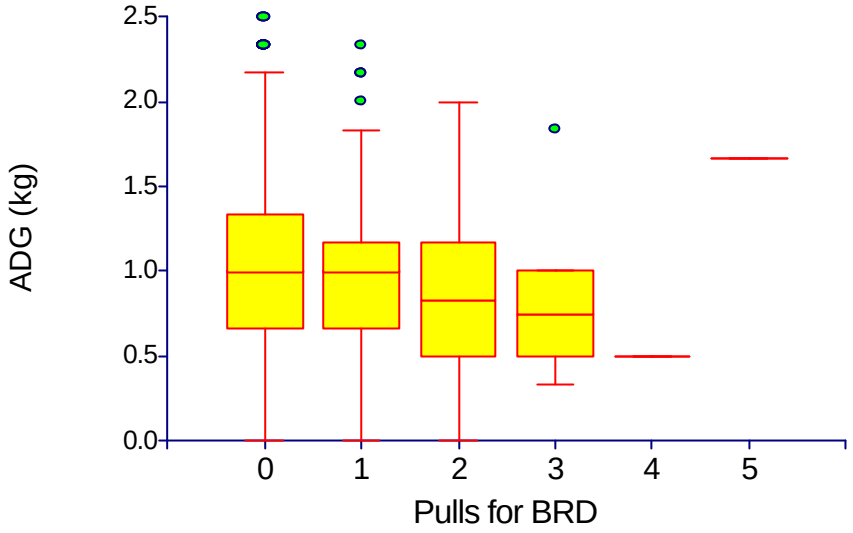

Figure 3. Effect of the number of BRD pulls on ADG from processing to Day 35

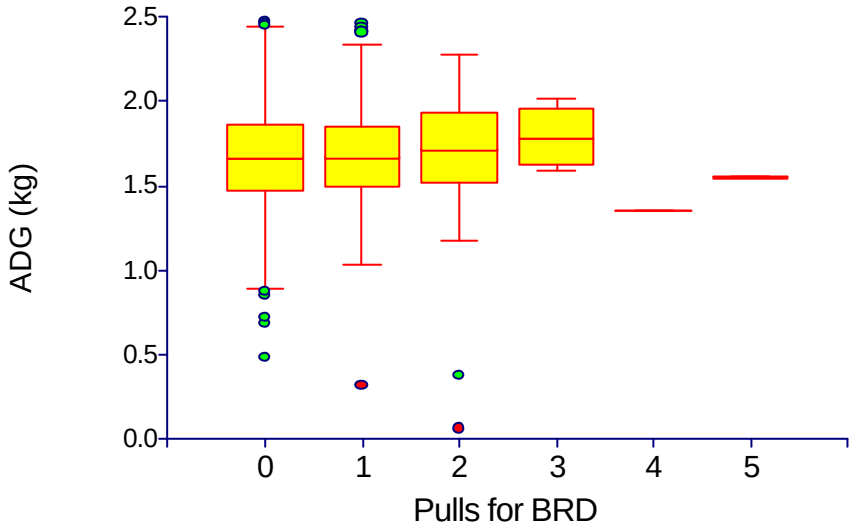

Figure 4. Effect of the number of BRD pulls on ADG from Day 35 to slaughter

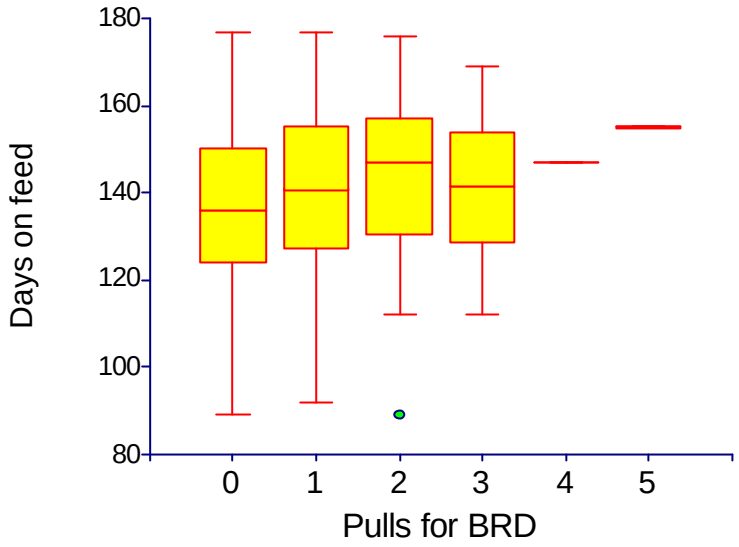

Figure 5. Effect of the number of BRD pulls on days on feed

5.3.2.2 Effect of lung lesions on performance

The presence of lung lesions (bronchopneumonia or adhesions/pleuritis) at slaughter was associated with a decrease in ADG from processing to slaughter of 27 g/day (P = 0.007) (Table 12). From processing to Day 35 a decrease of 89 g resulted (P <0.001). The effect of presence of lung lesions at slaughter on DOF was an increase of 5.41 days (P <0.001) (Table 13).

Table 12. Final multiple regression models: effect of the presence of lung lesions at slaughter on average daily gain (ADG) in feedlot cattle

Outcome	Predictor and level	*b*	S.E.(*b*)	95% confidence interval	*P*-value
ADG from processing to slaughter	Lung lesions present				
	Yes	-0.027	0.010	-0.046, -0.007	0.007
	No	0[a]	0	-	-
	Processing mass				
	Continuous	0.0008	0.0002	0.0005, 0.001	<0.001
	Sex				
	Male	0.265	0.013	0.239, 0.291	<0.001
	Female	0[a]	0	-	-
	Anaplasmosis				
	Yes	-0.060	0.022	-0.103, -0.018	0.005
	No	0[a]	0	-	-
	Origin				
	k=21	-	-	-	<0.001
ADG from processing to Day 35	Lung lesions present				
	Yes	-0.089	0.023	-0.134, -0.044	<0.001
	No	0[a]	0	-	-
	Sex				
	Male	0.224	0.029	0.167, 0.281	<0.001
	Female	0[a]	0	-	-
	Origin				
	k=21	-	-	-	<0.001
ADG from Day 35 to slaughter	Lung lesions present				
	Yes	-0.017	0.012	-0.041, 0.007	0.15
	No	0[a]	0	-	-
	Processing mass				
	Continuous	0.001	0.0002	0.0006, 0.001	<0.001
	Sex				
	Male	0.261	0.016	0.230, 0.293	<0.001
	Female	0[a]	0	-	-
	Anaplasmosis				
	Yes	-0.096	0.026	-0.147, -0.044	<0.001
	No	0[a]	0	-	-
	Origin				
	k=21	-	-	-	<0.001

[a] Reference level

Table 13. Final multiple regression model: effect of the presence of lung lesions at slaughter on days on feed (DOF) in feedlot cattle

Predictor and level	*b*	S.E.(*b*)	95% confidence interval	*P*-value
Lung lesions present				
Yes	5.41	0.79	3.86, 6.96	<0.001
No	0[a]	0	-	-
Processing mass				
Continuous	-0.09	0.01	-0.11, -0.06	<0.001
Sex				
Male	5.92	1.05	3.87, 7.98	<0.001
Female	0[a]	0	-	-
Anaplasmosis				
Yes	4.57	1.71	1.22, 7.92	0.008
No	0[a]	0	-	-
Origin				
k=21	-	-	-	<0.001

[a] Reference level

5.3.2.3 Effect of bronchopneumonia score on performance

The decreases in ADG associated with increasing bronchopneumonia score were only marginally significant (Table 14), except for the effect of score 2 vs. score 0, which resulted in a 88 g/day reduction in ADG after Day 35 (P = 0.02). No significant effect of bronchopneumonia score on DOF was found (Table 15).

Table 14. Final multiple regression models: effect of bronchopneumonia score at slaughter on average daily gain (ADG) in feedlot cattle

Outcome	Predictor and level	*b*	S.E.(*b*)	95% confidence interval	*P*-value
ADG from processing to slaughter	Bronchopneumonia score				
	0	0[a]	0	-	-
	1	-0.035	0.021	-0.076, 0.006	0.09
	2	-0.046	0.030	-0.104, 0.013	0.1
	Processing mass				
	Continuous	0.0008	0.0002	0.0005, 0.001	<0.001
	Sex				
	Male	0.264	0.013	0.238, 0.290	<0.001
	Female	0[a]	0	-	-
	Anaplasmosis				
	Yes	-0.061	0.022	-0.103, -0.019	0.005
	No	0[a]	0	-	-
	Origin				
	k=21	-	-	-	<0.001
ADG from processing to Day 35	Bronchopneumonia score				
	0	0[a]	0	-	-
	1	-0.068	0.049	-0.164, 0.027	0.16
	2	0.064	0.069	-0.071, 0.199	0.4
	Sex				
	Male	0.224	0.029	0.166, 0.281	<0.001
	Female	0[a]	0	-	-
	Origin				
	k=21	-	-	-	<0.001
ADG from Day 35 to slaughter	Bronchopneumonia score				
	0	0[a]	0	-	-
	1	-0.025	0.026	-0.076, 0.026	0.3
	2	-0.088	0.036	-0.160, -0.017	0.02
	Processing mass				
	Continuous	0.001	0.0002	0.0007, 0.001	<0.001
	Sex				
	Male	0.260	0.016	0.229, 0.292	<0.001
	Female	0[a]	0	-	-
	Anaplasmosis				
	Yes	-0.097	0.026	-0.148, -0.046	<0.001
	No	0[a]	0	-	-
	Origin				
	k=21	-	-	-	<0.001

[a] Reference level

Table 15. Final multiple regression model: effect of bronchopneumonia score at slaughter on days on feed (DOF) in feedlot cattle

Predictor and level	*b*	S.E.(*b*)	95% confidence interval	*P*-value
Bronchopneumonia score				
0	0[a]	0	-	-
1	-1.30	1.68	-4.60, 2.00	0.4
2	-0.55	2.40	-5.25, 4.15	0.8
Processing mass				
Continuous	-0.09	0.01	-0.11, -0.06	<0.001
Sex				
Male	6.01	1.06	3.94, 8.09	<0.001
Female	0[a]	0	-	-
Anaplasmosis				
Yes	4.55	1.73	1.16, 7.94	0.009
No	0[a]	0	-	-
Origin				
k=21	-	-	-	<0.001

[a] Reference level

5.3.2.4 Effect of adhesions/pleuritis on performance

Adhesions and/or pleuritis was strongly associated with a decrease in ADG. This was most outspoken for the period from processing to Day 35 (Table 16). A score of one showed an increase of 6.39 days on feed (*P* <0.001) (Table 17).

Table 16. Final multiple regression models: effect of pleural adhesion score at slaughter on average daily gain (ADG) in feedlot cattle

Outcome	Predictor and level	*b*	S.E.(*b*)	95% confidence interval	*P*-value
ADG from processing to slaughter	Adhesion score				
	0	0[a]	0	-	-
	1	-0.024	0.010	-0.044, -0.004	0.02
	2	-0.088	0.038	-0.163, -0.014	0.02
	Processing mass				
	Continuous	0.0008	0.0002	0.0004, 0.001	<0.001
	Sex				
	Male	0.265	0.013	0.239, 0.291	<0.001
	Female	0[a]	0	-	-
	Anaplasmosis				
	Yes	-0.061	0.022	-0.103, -0.019	0.005
	No	0[a]	0	-	-
	Origin				
	k=21	-	-	-	<0.001
ADG from processing to Day 35	Adhesion score				
	0	0[a]	0	-	-
	1	-0.103	0.024	-0.149, -0.057	<0.001
	2	-0.227	0.087	-0.397, -0.057	0.009
	Sex				
	Male	0.224	0.029	0.167, 0.281	<0.001
	Female	0[a]	0	-	-
	Origin				
	k=21	-	-	-	<0.001
ADG from Day 35 to slaughter	Adhesion score				
	0	0[a]	0	-	-
	1	-0.011	0.013	-0.036, 0.014	0.4
	2	-0.053	0.046	-0.144, 0.037	0.2
	Processing mass				
	Continuous	0.001	0.0002	0.0006, 0.001	<0.001
	Sex				
	Male	0.261	0.016	0.229, 0.293	<0.001
	Female	0[a]	0	-	-
	Anaplasmosis				
	Yes	-0.096	0.026	-0.148, -0.045	<0.001
	No	0[a]	0	-	-
	Origin				
	k=21	-	-	-	<0.001

[a] Reference level

Table 17. Final multiple regression model: effect of pleural adhesion score at slaughter on days on feed (DOF) in feedlot cattle

Predictor and level	*b*	S.E.(*b*)	95% confidence interval	*P*-value
Adhesion score				
0	0[a]	0	-	-
1	6.39	0.81	4.80, 7.98	<0.001
2	1.27	3.01	-4.64, 7.17	0.7
Processing mass				
Continuous	-0.08	0.01	-0.11, -0.06	<0.001
Sex				
Male	5.68	1.05	3.63, 7.73	<0.001
Female	0[a]	0	-	-
Anaplasmosis				
Yes	4.56	1.70	1.22, 7.90	0.008
No	0[a]	0	-	-
Origin				
k=21	-	-	-	<0.001

[a] Reference level

5.3.2.5 Effect of respiratory disease on performance

The overall effect of BRD (defined as an animal diagnosed with clinical BRD and/or an animal with lung lesions present at slaughter) on performance was a decrease in ADG of 28 g from processing to slaughter ($P = 0.06$). A 144 g/day decrease was shown from processing to Day 35 ($P < 0.001$) but no significant difference from Day 35 to slaughter (Table 18). An increase of 4.95 days on feed was shown for animals with respiratory disease ($P < 0.001$) (Table 19).

Table 18. Final multiple regression models: effect of respiratory disease (pulled for respiratory disease and/or lung lesions present at slaughter) on average daily gain (ADG) in feedlot cattle

Outcome	Predictor and level	*b*	S.E.(*b*)	95% confidence interval	*P*-value
ADG from processing to slaughter	Respiratory disease				
	Yes	-0.028	0.010	-0.047, -0.008	0.006
	No	0[a]	0	-	-
	Processing mass				
	Continuous	0.0008	0.0002	0.0005, 0.001	<0.001
	Sex				
	Male	0.266	0.013	0.240, 0.291	<0.001
	Female	0[a]	0	-	-
	Anaplasmosis				
	Yes	-0.059	0.022	-0.102, -0.017	0.006
	No	0[a]	0	-	-
	Origin				
	k=21	-	-	-	<0.001
ADG from processing to Day 35	Respiratory disease				
	Yes	-0.144	0.023	-0.188, -0.099	<0.001
	No	0[a]	0	-	-
	Sex				
	Male	0.228	0.029	0.171, 0.284	<0.001
	Female	0[a]	0	-	-
	Origin				
	k=21	-	-	-	<0.001
ADG from Day 35 to slaughter	Respiratory disease				
	Yes	0.000	0.012	-0.024, 0.024	0.998
	No	0[a]	0	-	-
	Processing mass				
	Continuous	0.001	0.0002	0.0006, 0.001	<0.001
	Sex				
	Male	0.261	0.016	0.229, 0.293	<0.001
	Female	0[a]	0	-	-
	Anaplasmosis				
	Yes	-0.096	0.026	-0.147, -0.044	<0.001
	No	0[a]	0	-	-
	Origin				
	k=21	-	-	-	<0.001

[a] Reference level

Table 19. Final multiple regression model: effect of respiratory disease (pulled for respiratory disease and/or lung lesions present at slaughter) on days on feed (DOF) in feedlot cattle

Predictor and level	*b*	S.E.(*b*)	95% confidence interval	*P*-value
Respiratory disease				
Yes	4.95	0.79	3.40, 6.51	<0.001
No	0[a]	0	-	-
Processing mass				
Continuous	-0.09	0.01	-0.11, -0.06	<0.001
Sex				
Male	5.84	1.05	3.78, 7.90	<0.001
Female	0[a]	0	-	-
Anaplasmosis				
Yes	4.39	1.71	1.03, 7.75	0.01
No	0[a]	0	-	-
Origin				
k=21	-	-	-	<0.001

[a] Reference level

5.4 Economic analysis

The economic analysis was done only to include variable costs. Direct variable costs were treatment costs taken at list drug prices for April 2003 [48], and labour costs estimated to be around R2 per animal per treatment course.

5.4.1 Direct variable costs

Direct variable cost per animal entering the feedlot was calculated as follows:

= (Total treatment costs + labour costs + mortality cost) ¸ total number of animals entering feedlot

= R 31 354.40 ¸ 2036

= R 15.40 / animal entering the feedlot

5.4.2 Indirect or hidden variable costs

Indirect variable cost was due to loss of production resulting from BRD and was calculated as follows:

= Reduction in ADG due to BRD ´ mean DOF ´ mean dressing percentage ´ meat price ´ incidence of BRD

= 0.028 kg/day ´ 136 ´ 58% ´ R13 /kg ´ 52%

= R 14.93 / animal entering the feedlot

The total cost of BRD was the sum of direct variable costs and indirect variable costs, i.e. R30.33/ animal entering the feedlot.

6. Discussion

The treatment rate for respiratory disease in this study was 22.7%. This is within the range of 15 –50% reported in other studies [2,5,17,59,60]. The variations in findings of the different studies could be due to a variety of factors. Stressors, origin of calves (farm vs. auction), severity of BRD challenge, and management will be different for every study and will affect the incidence of BRD. Bovine Respiratory Disease was the most common disease found in the study animals.

The peak incidence of BRD on Day 18 is consistent with findings of other studies. Bateman *et al.* [2] found the median day of first treatment 10 days after arrival, and Wilson *et al.* [59] found the highest incidence on day 30 after arrival. Management in the first 5 - 7 weeks after arrival in the feedlot is critical as this is clearly the period of highest risk for BRD.

The prevalence of lung lesions (bronchopneumonia and/or adhesions) was 42.7%. Other studies reported 72% [60], 42% [5] and 74% [17]. Of animals treated for BRD, 57% still had lung lesions at slaughter. These animals may have been treated late, the treatment may not have been completely effective or alternatively some animals may have experienced another, undetected episode of BRD either before or during the feeding period. The use of anti-inflammatory drugs could also have had an effect on the outcome possibly limiting lung lesions especially adhesions and pleuritis. Because Flunixin meglumine was used in animals with more clinically severe disease, and not randomly, it was not possible to take into account its effect on performance in an unbiased manner in this study.

Of animals never treated for clinical respiratory disease, 38.5 % had lung lesions at slaughter. Other studies reported 37%[17], 42%, [5] and 68% [60]. Of animals with lung lesions at slaughter, nearly 70% had never been diagnosed with clinical respiratory disease. Wittum *et al.* [60] found this to be 68% and Gardner *et al.* [17], 37%. This shows that the association between treatment for BRD and lung lesions at slaughter is poor. A lot of cases are missed, indicating a low sensitivity using treatment records as the only way of estimating the incidence of disease.

Using the combined case definition of treated animals and/or lung lesions present at slaughter, the estimated incidence of BRD during this study was 52%. The sensitivity for detecting BRD using this case definition is higher than when using either treatment records or presence of lung lesions alone, because false negatives are fewer. However, the sensitivity using the combined case definition is not perfect, as subclinical BRD and even some clinical cases, not severe enough to cause persistent lung lesions, will still be missed.

Using this combined case definition, however, will lower the specificity of the diagnosis, as pre-existing cases and other diseases misdiagnosed as BRD (false positives), will also be included in the BRD group. Nevertheless using the combined case definition is probably a more accurate estimate of the true incidence of BRD in feedlots.

The starting mass from which ADG was calculated in this study was processing mass, rather than arrival mass. The interval from arrival to processing was 5 days and mass differences of individual animals changed by as much as 10 kg over this period. This could probably be the effect of differences in rumen fill and hydration status between arrival and processing. We decided to use the three weights (processing weight, Day 35 weight and slaughter weight) as these weights were recorded as part of the normal protocol in the feedlots. This enabled us to calculate ADG for an early period and a later period in the feedlot and from this we could determine more precisely when each predictor used was influencing the animal's growth.

In this study it was found that treatment for BRD resulted in an overall decrease in ADG of 19 g for the whole period in the feedlot. This decrease was most outspoken in the first 30 days (165 g/day), after which no further loss in ADG occurred. After Day 35 there was a tendency for treated animals to grow slightly faster, which may indicate that a small amount of compensatory growth occurred. However, this was not statistically significant, nor was it sufficient to offset the earlier losses. Other studies found a decrease of 60 g/day for the whole feeding period [2,17]. One study, that only measured ADG from arrival to Day 28 in the feedlot, found a decrease of 140 g/day for treated animals [51]. Some studies however, have found no association between treatment for BRD and ADG [24,60]. These discrepancies between studies regarding the impact of morbidity on performance may be due to several factors. The case definition used for BRD may be different between studies.

In some studies a fever had to be present for a diagnosis of BRD [24], whilst others took the animal's clinical signs together with temperature measurements into account [2,17]. This means that for some studies only the more severe cases, with longer recovery times, may have been identified which may have biased results. Viral causes of BRD with no secondary bacterial involvement, causing no lesions in the respiratory tract, may affect ADG much less although such cases may still be pulled. The accuracy of clinical diagnosis of bovine respiratory disease, as well as the severity of the disease, may vary greatly between feedlots and this will also influence the impact of BRD on ADG.

No significant differences in ADG were found between animals treated once and those treated more than once. In a 90 day Canadian trial, retreated animals had decreased gains of nearly twice that of animals treated once only [36]. In a 150 day trial a decreased gain of 21 kg was found for animals treated more than once vs. animals treated once only [17]. Although a significant effect of re-treatment was shown in other studies, there was insufficient evidence in this study to support this. In our study, however, we found that retreated animals had a greater chance of having adhesions/pleuritis at slaughter than animals treated once only (64% vs. 52%; P =0.05). Re-treatments may thus have an indirect negative effect on ADG through being associated with adhesions.

The presence of lung lesions (bronchopneumonia and/or adhesions/pleuritis) at slaughter was associated with a decrease in ADG of 27 g for the period from processing to slaughter. This is similar to findings by Bryant (26 g) [5], but less than that reported by Gardner (180 g) [17] and Wittum (76 g) [60]. The difference between studies in the effect of lung lesions on ADG could be due to different methods of lung lesion scoring or differences in severity of respiratory disease.

The largest and most significant effect of bronchopneumonia score on ADG was for severe bronchopneumonia (score 2 vs. score 0) from Day 35 to slaughter (88 g/day) and indeed is the only respiratory parameter to significantly affect growth during this period. It may be that severe pneumonia is more likely to be overlooked during this period in the feedlot, possibly due to less attention being given to respiratory disease at this time.

In contrast, the presence of adhesions was very strongly associated with a reduction in ADG during the first 35 days (103 g/day, score 1; 227 g/day, score 2). It seems that severe

inflammation in the early feeding period, extensive enough to cause persistent adhesions/pleuritis, has a negative effect on ADG at the time, but in general does not have a significant effect later on in the feeding period.

Possible mechanisms for reduced performance in calves suffering from BRD could be reduced feed intake, reduced feed conversion ratio (FCR) and catabolic events (fever accelerating protein and energy metabolism) [22] associated with BRD. Unfortunately, individual FCR was impossible to determine because individual feed intake was not measured. Reduced intake may result in a saving on feed, but the longer DOF required to reach market mass results in a reduced efficiency of the feedlot operation as a whole.

The presence of lung lesions is more strongly associated with reduced ADG than the occurrence of clinical disease, presumably because it can be assumed that only severe BRD will cause lung lesions. Lung lesions may also persist long after resolution of clinical disease. If less surface area in the lungs is available for exchange of oxygen it is possible that oxygen levels in the blood may be lower, negatively affecting all anabolic processes in the body.

The estimate of 52% incidence of BRD is much higher than the clinical incidence of BRD (23%) or the prevalence of lung lesions (43%) at slaughter and is likely to be closer to the true incidence of BRD during the feeding period. This enabled a more accurate calculation of the overall effect of BRD on the performance of feedlot cattle and the economic losses associated with BRD suffered by feedlot owners in South Africa. The study found that the occurrence of BRD was associated with a decrease in ADG of 144 g for the period from processing to Day 35 and an overall decrease of 28 g for the period from processing to slaughter. For an average standing time of 136 days in the feedlot, this translated into a hidden cost of R14.93 per animal in the feedlot. This was nearly equal to the cost of medicines and labour. The total loss due to BRD was estimated to be R30.30 per animal in the feedlot.

No mortalities occurred due to BRD during this period, but mortalities would obviously increase known costs. List drug prices were used in the calculations and bulk discounts for which feedlots can qualify was not included; in reality, therefore, treatment costs are likely

to be lower. It follows that the hidden costs of BRD resulting from the decreased growth probably exceeds the known costs of treatment.

The approximate throughput of cattle in all South African feedlots is 1.35 million animals per annum [1]. If this is multiplied by a cost of R30 per animal it translates to a cost of about R40m per year to the feedlot industry. This does not include costs of preventive measures (vaccines and chemoprophylaxis) and mortalities due to BRD

We believe that the feedlots used in the study are representative of typical feedlots in South Africa and all factors affecting feedlot diseases were present at the selected feedlots. However, deep pulling (animals were selected to be treated at the earliest signs of respiratory disease) of animals was done, resulting in low mortalities in the feedlots. This is perhaps not the case at all feedlots in South Africa. Obviously, in feedlots where management is poor, pulling is late or inaccurate and mortalities due to BRD are high, expected costs of BRD can be much higher.

It was found in this study that missed and subclinical cases of BRD are also very commonplace in South African feedlots as in other parts of the world. It could be beneficial to consider the inclusion of prophylactic antimicrobials for higher risk animals to reduce the rate of subclinical infections, the more common usage of anti-inflammatory drugs to prevent severe inflammation with the formation of adhesions and pleuritis, and to ensure that pulling and treatment of animals is done as accurately as practically possible. More attention should also be given to identification and treatment of animals with BRD after day 35 in the feedlot. Considering the direct and indirect costs of BRD in the feedlot, it may be advisable to invest more resources into buying calves that have a lower risk of suffering from BRD. This could include employment of practices such as preconditioning and backgrounding. High-risk groups of cattle should be avoided unless the lower price paid for these calves will offset the known and hidden costs of BRD.

With the findings of this study as a guideline, feedlot owners will be able to take more realistic account of the true costs associated with respiratory disease in their operations. Future studies should look at quantifying the costs of prevention vs. the costs of disease in order to formulate cost effective preventive measures against BRD.

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