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# The medicine and epidemiology of bovine respiratory disease in feedlots

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Bovine Respiratory Disease (BRD) results from a complex, multifactorial interaction of stressors, animal susceptibility, and respiratory pathogens. The infectious agents associated with BRD are ubiquitous among cattle populations. Typically, one or a combination of stressors are necessary to initiate BRD. Prevention of BRD should, therefore, address management procedures to minimise stressors. Administration of vaccines against BRD agents may help reduce the incidence of BRD but is unlikely to eliminate the condition. The effectiveness of antimicrobials in the treatment of BRD depends primarily on early recognition and treatment. The use of antioxidant vitamins, minerals or other agents in the prevention and treatment of BRD warrants further research.
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|---|---|
| BHV1 | Bovine herpesvirus 1 |
| BRD | Bovine respiratory disease |
| BRSV | Bovine respiratory syncytial virus |
| BVDV | Bovine viral diarrhoea virus |
| IBR | Infectious bovine rhinotracheitis |
| NSAIDs | Non steroidal anti-inflammatory drugs |
| PI-3 | Parainfluenza virus 3 |

The aim of this review is to examine the information available on the causes, treatment and prevention of BRD. This epidemiological approach to BRD management allows assessment of the clinical application of existing and potential interventions. All journals and proceedings with English translations were searched electronically on the topic of BRD and the abstracting service of Hoffmann-La Roche, Australia, was used.

## Factors which predispose feedlot cattle to bovine respiratory disease

*Transport and time without feed* — Fasting and transport are factors that predispose cattle to the development of BRD by causing immunosuppression and increasing cellular oxidative challenge.[1] Transport impairs calves' immune responses as measured by lymphocyte blastogenesis,[2] and may lengthen the time required for restoration of ruminal volume and volatile fatty acid production after a fasting period, perhaps due to the increased water loss associated with transport in addition to water deprivation alone.[3] Transport can exacerbate the effects of fasting through increased social interaction between calves. Calves held in individual stalls in a moving vehicle had approximately half the weight loss of calves transported while in physical contact with each other.[4] Weight and nutrient losses were also approximately 50% lower in fasted calves compared with calves fasted and transported.[4] Transport can result in irritation of airways subjected to prolonged exposure to exhaust fumes. When the exhaust stack on the prime-mover was lower than the top of the trailer, calves that travelled on the top deck tended to have lower subsequent feedlot growth rates than calves that travelled on the lower deck.[5] Conversely, calves from the top deck had higher feedlot growth rates than calves from the bottom deck when the exhaust stack was higher than the trailer.[5]

Most of the stress of transport of less than 24 h duration appears to be related to the loading and unloading process.[6] For trips of 24 h or less, transport distance was not found to be directly related to subsequent health and performance.[6] Conversely, trans-

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port periods greater than 24 h were associated with a greater incidence of BRD.[7]

*Mixing cattle from different sources* — Morbidity and mortality from BRD increase with mixing of calves from different sources and assembly of calves from widely separated geographic locations.[8] The bacteria commonly isolated from clinical BRD (*Mannheimia haemolytica* and *Pasteurella multocida*) have been isolated from the upper and lower airways of both clinically affected and healthy cattle.[9] Furthermore, the infectious agents associated with BRD are ubiquitous in the cattle population,[10] and the occurrence of BRD is not distributed evenly across a pen.[11] Increased morbidity and mortality from BRD therefore appears not to be caused solely by infectious challenge encountered by naïve cattle mixed with cattle previously exposed to BRD infectious agents, but also by the stress of establishment of a new social heirachy.[12] Australian cattle maintained as a group from weaning until feedlot entry adapted more rapidly to the feedlot ration and had higher growth rates over the first 37 days compared with cattle purchased through saleyards from a variety of sources.[13]

*Introductory diet* — There is a strong association between feeding corn silage during the first month in the feedlot and increased incidence of BRD.[8] In the Bruce County Beef Project's analysis of introductory feeding practices, mortality due to BRD was five times higher in calves fed corn silage as a major portion of their diet during the first week in the feedlot than in calves that were not fed substantial amounts of corn silage until the fourth week. Feeding grain with the silage appeared to reduce some of the negative effects of silage consumption. Inclusion of non-protein nitrogen in the introductory diet in addition to that in the silage was also associated with increased mortality. Although analyses of the diets were not provided in this study it appears that feeding excessive amounts of non-protein nitrogen with inadequate rumen degradable true protein and inadequate starch and sugars may be responsible for the observed increase in the incidence of BRD rather than silage feeding per se. Another study showed a reduction in morbidity and mortality when newly arrived calves were fed grass hay only, but this feeding practice resulted in a decrease in growth rate.[14] If hay was provided for longer than 3 days in the receiving pen, it tended to inhibit intake of mixed ration, thereby reducing energy intake in newly arrived cattle.[7] It is possible that inappropriate ration formulation may be a risk factor for the development of BRD, most likely effected by depressed energy and true protein intake by cattle in a catabolic state.

Cattle purchased in saleyards and introduced to diets containing 20 to 30% high moisture barley were 4.9 times more likely to be treated for BRD, and 6.7 times more likely to die from BRD, than cattle assembled on their farm of origin and started on a diet containing 10% high moisture barley.[15] Conversely, cattle with low blood glucose concentrations on arrival at the feedlot had a greater chance of subsequently developing severe BRD, however, morbidity and mortality were reduced in calves fed a diet containing 55% concentrate rather than good quality hay at the saleyards before transport to the feedlot.[16] Although rumen pH was not measured in these studies, the effects of higher grain diets on the incidence of BRD may be mediated by the development of lactic acidosis, a disorder which is influenced by feed milling and delivery in addition to diet formulation. It may be that diets with at least 50% concentrates can reduce the incidence of BRD in cattle newly arrived at the feedlot provided they do not result in lactic acidosis. The appropriate formulation of the initial diet for cattle on arrival at feedlots requires further research.

*Climate* — The peak incidence of BRD usually occurs in autumn and early winter in Australia and the USA.[17] Whereas the association between season and BRD incidence in the USA could be confounded by the influx of light weight calves in autumn, feedlot cattle numbers do not consistently vary with season in eastern Australia. More rapid and severe temperature changes and greater weather extremes in the USA contribute to higher BRD morbidity and mortality rates compared with Australia.[17] It appears that rapid change in temperature, rather than temperature per se, is responsible for an increase in the incidence of BRD.

## The aetiology and pathophysiology of BRD

*Introduction* — Various systems of nomenclature have been developed over recent decades to describe specific conditions belonging to BRD.[18] They describe a suite of conditions that are influenced by one or many stressors that result in sufficient immunocompromisation to allow pulmonary invasion by viral and/or bacterial pathogens that are ubiquitous in the feedlot environment. Differentiation between the lesions caused by various BRD agents is largely irrelevant to the prevention of BRD in feedlots. Efforts to reduce or eliminate stressors that provide an opportunity for colonisation of the lungs by pathogens reduces the incidence of BRD.[19]

*Viral respiratory pathogens* — Frequently, bacterial pneumonia is preceded by a viral respiratory infection. Many cattle entering Australian feedlots have antibodies against the viruses most commonly implicated in the pathogenesis of BRD.[20] A 1991 survey of 233,450 cattle on arrival at six feedlots found 68, 13, 57 and 27% were serologically positive for BVDV, BHV1, PI-3 and BRSV respectively.[20] Of the cattle that were serologically negative to these viruses at feedlot entry, retesting at slaughter found 94, 76, 78 and 71% were now positive for BVDV, BHV1, PI-3 and BRSV respectively.[20] While in the feedlot 6.8% of the cattle required treatment, 0.9% died, and 53% of the deaths were due to BRD. Clinical signs of disease were observed in only 10.3% of the cattle that serologically converted to one or more respiratory viruses. Even though viral infection is a risk factor for BRD, viral respiratory infection alone is not sufficient to cause BRD.

*Bovine herpesvirus 1* — BHV1 is the aetiological agent responsible for IBR. Signs and lesions range from serous, hyperaemic and oedematous membranes, through mucopurulent exudate with focal necrosis, to pseudomembranous inflammation in severe cases.[21] Latent infection with BHV1 can occur in the trigeminal ganglia and stress may precipitate recrudescence of the virus with clinical signs and viral shedding.[21]

BHV1 replicates in mucosal cells[22] and in various cell types of the submucosa and connective tissue peripheral to the tracheal rings.[18] This may lead to destruction of the epithelium of the upper respiratory tract[22] with cessation of ciliary activity resulting in loss of function of the mucociliary escalator.[23] Consequently, secondary bronchopneumonia may occur due to inhalation of infectious tracheal exudates and failure to clear particulate fomites and bacteria from the lungs.[18] Field observations support this as a frequent sequel to IBR, as a high proportion of cattle at postmortem following BRD with IBR have an anteroventral lobar distribution of lesions resembling inhalation pneumonia. BHV1 can also cause excessive bronchoconstric-

tion resulting in trapping of secretions in the lower airways, thereby impairing lung defence mechanisms and favouring bacterial growth.[23] BHV1 infection causes immunosuppression that can increase susceptibility to secondary bacterial infections resulting in severe pneumonia.[24] This immunosuppression may reduce neutrophil migration, cell-mediated cytotoxicity, mitogen responses of peripheral blood lymphocytes, and some functional activities of alveolar macrophages.[25]

*Parainfluenza virus type 3* — Following experimental infection, PI-3 replicates in epithelial cells of both the upper and lower respiratory tract. However, damage occurs primarily in the lower respiratory tract. Viral replication in the epithelial cells of the lower respiratory tract causes bronchitis, bronchiolitis and alveolitis.[26] In the acute stage of PI-3 virus infection, there is proliferation and necrosis of bronchiolar epithelial cells with widespread destruction of cilia and of ciliated cells in small bronchi and bronchioli.[27] PI-3 infects alveolar macrophages[26] and thereby impairs innate pulmonary defence mechanisms. Thus, cattle may be predisposed to secondary bacterial pneumonia because of suboptimal mucociliary escalator function and depressed local cellular immune responses.

*Bovine viral diarrhoea virus* — The role of BVDV in the pathogenesis of BRD has been subject to much conjecture due to a lack of evidence implicating it as a primary BRD pathogen. BVDV may facilitate colonisation of the lungs by other pathogens.[28] Experimental infection of immunocompetent, seronegative calves with BVDV type 1d induced primary BRD, in the absence of concurrent infection with other BRD pathogens,[29] suggesting a possible primary role for the virus in the pathogenesis of BRD.

The immunosuppressive effect of acute BVDV infection appears to be mediated by initial hyperplasia of the germinal centres of all lymphoid organs within 10 days of infection, followed by lymphoid depletion.[29] In addition, BVDV impairs humoral antibody production, depresses monocyte chemotaxis and impairs the myeloperoxidase antibacterial system in polymorphonuclear leukocytes.[30] Presumably, these mechanisms enhance colonisation of the lungs by other BRD pathogens and exacerbate the pulmonary pathology they generate. The direct cytopathic effects of BVDV in the airways result in acute cattarrhal inflammation in the nasal cavity and trachea, and focal intralobular interstitial pneumonia.[29] It appears, therefore, that BVDV may enhance the development of BRD by immunosuppression and as a primary respiratory pathogen.

*Bovine respiratory syncytial virus* -— In common with the major viral respiratory disease agents, BHV1 and PI-3, BRSV infection results in destruction of the ciliated respiratory epithelium[31] and infection of alveolar macrophages depresses local cellular immunity.[26] This interference with pulmonary clearance predisposes cattle to secondary bacterial pulmonary infection.[26] Whereas involvement of BRSV in outbreaks of clinical respiratory disease has been reported in Europe and North America,[31] BRSV does not appear to play a major role in BRD in Australian feedlots based on the results of virus isolation from sick animals and postmortems.[32]

*Bacterial respiratory pathogens* — The 1991-1993 survey of diseases in eastern Australian feedlots[20] found that almost two thirds of feedlot treatments were attributable to BRD. However, bacteria were only cultured from 19% of sick animals. *Mannheimia haemolytica* (formerly known as *Pasteurella haemolytica*) was cultured from sick cattle more frequently than *Pasteurella multocida* (7% versus 2.7%). Of the deaths investigated, 53% were attributed to BRD. *Pasteurella multocida* was more commonly cultured from postmortem material than *M haemolytica* (14% versus 9%). Other bacteria commonly isolated were *Salmonella* spp (6%) and *Actinomyces pyogenes* (10%). *Haemophilus somnus* was only cultured from 2% of deaths, but this may reflect the fastidiousness of the organism in culture. Fourteen percent of postmortem cultures yielded various other bacteria. This survey clearly illustrates that while *M haemolytica* and *P multocida* are common causes of bacterial BRD, several species of bacteria can fill this niche if the opportunity presents. A similar observation has been made with BRD in North American cattle.[33]

*Mannheimia haemolytica and Pasteurella multocida* — Once bacteria are established in the lung, tissue damage is mediated by several mechanisms. Toxins play a major role in the pathogenesis of bacterial pneumonia. Lipopolysaccharide endotoxin in the outer membrane of Gram negative cell walls is involved in bacterial lung damage. Endotoxin has an array of toxic effects including initiation of complement and coagulation cascades.[34] These result in increased vascular permeability and coagulation leading to accumulation of inflammatory cells, oedema and both intravascular and extravascular fibrin deposition in the lung.[34] Endotoxin activates granulocytes and macrophages that help protect against bacteria that contain endotoxins but also lead to increased tissue damage. *M haemolytica* produces a ruminant specific leukotoxin active against phagocytes that impairs phagocytosis and kills macrophages.[34] *M haemolytica* and *P multocida* can also affect neutrophil defence of the lung. Extracellular fractions of *M haemolytica* kill bovine neutrophils[35] and a capsular fraction of *P multocida* inhibits bovine neutrophil function.[34] These bacteria, therefore, attract phagocytes into the affected regions of the lungs and destroy the phagocytes using toxins. The reactive oxygen metabolite contents of the phagocytes, which were otherwise destined to destroy phagocytosed bacteria in phagolysosomes, are consequently released. In this way, much of the damage in bacterial pneumonia involving *M haemolytica* and *P multocida* is due to pulmonary inflammation.

*Haemophilus somnus* — Like *M haemolytica* and *P multocida*, *H somnus* has a lipopolysaccharide endotoxin in the outer membrane of its Gram negative cell wall. This endotoxin causes similar lesions to those produced by the lipopolysaccharide of *M haemolytica* and *P multocida*, but also causes vasculitis and necrosis.[34] *Haemophilus somnus* also elaborates exotoxins that damage endothelial cells,[36] alveolar macrophages and neutrophils.[34] These factors are presumably important in the pathogenesis of pulmonary disease due to *H somnus* infection causing vasculitis, consequent attraction of phagocytic cells and destruction of these with release of their oxyradical contents.

## Conclusions on the epidemiology of bovine respiratory disease in feedlot cattle

The major pathogens of BRD are ubiquitous[10] and all the major bacterial respiratory pathogens are commensal in clinically normal feedlot age cattle.[33] Clinical BRD is a product of the effects of stressors causing immunosuppression, which allows colonisation of the respiratory tract by opportunistic pathogens inevitably encountered by feedlot cattle. Infectious challenge only plays a minor role in the development of BRD.[11] It is therefore inappropriate to describe major BRD events as outbreaks, rather, it is more accurate to describe them as disease incidence spikes that occur in response to a combination of

sufficient stressors. Attempts to reduce the incidence of BRD by isolation of clinical cases will be of limited value. Control of BRD is most effectively achieved by minimising the stressors responsible for making cattle susceptible to clinical infections with organisms they are inevitably exposed to in the feedlot.

## Treatment of bovine respiratory disease

*Antimicrobials* - Antimicrobials are indicated in the treatment of cases of BRD involving primary or secondary bacterial infections. Antibiotics registered for use in cattle in Australia and commonly used to treat BRD include oxytetracycline, trimethoprim potentiated sulfonamides, tilmicosin and ceftiofur.

Oxytetracycline,[37] trimethoprim potentiated sulfonamides,[38] tilmicosin,[39] and ceftiofur[40] have all been shown to significantly reduce the severity of clinical signs of BRD, and to reduce the case fatality rate. Florfenicol is used to treat BRD in North America and it has been found to be effective in reducing relapse and case fatality rates.[41] Enrofloxacin has also been used to effectively treat BRD in the USA and Europe.[42]

Comparative studies have shown variation in the efficacy of antimicrobials in the treatment of BRD (Table 1). The efficacy of antimicrobials is affected by sensitivity of the target organisms and early recognition and treatment of cases. Resistance to the commonly used antimicrobials is rare in bacterial isolates from cattle with BRD in Australian feedlots (Taylor, unpublished data, 2000, table 2) compared with a relatively high frequency of antimicrobial resistance in North American BRD isolates.[48-50] This in vitro sensitivity finding is supported by high treatment success rates in cattle treated in Australian feedlots.[20] Antibiotic selection is therefore determined primarily by cost, duration of action, ease of administration, and length of withholding period from slaughter. It is possible that cattle with chronic pulmonary abscessation may respond better to an antibiotic more capable of penetrating necrotic tissue and pus such as trimethoprim/sulfonamide[51] but the prognosis with such cases is grave and financial considerations usually dictate salvage slaughter.

**Table 1. Comparative studies on the efficacy of antimicrobials for the treatment of bovine respiratory disease in North America.**

| Antimicrobials compared | Antimicrobial of greater efficacy | P - value | Outcome measured |
|---|---|---|---|
| Florfenicol and tilmicosin | Florfenicol[43] | < 0.05 | Mortality |
| | Tilmicosin[44] | < 0.02 | Treatment success[a] |
| | | < 0.01 | Weight gain |
| | No significant difference[45] | 0.20 | Mortality |
| | | 0.10 | Treatment success |
| Tilmicosin and oxytetracycline | Tilmicosin[37] | < 0.04 | Treatment success |
| | No significant difference[46] | Not quoted | Relapse rate |
| | | | Weight gain |
| Tilmicosin and trimethoprim/sulfonamide | Tilmicosin[37] | < 0.07 | Treatment success |
| Trimethoprim/sulfonamide, oxytetracycline and penicillin | Trimethoprim/sulfonamide[38] | < 0.05 | Treatment days per case |
| | No significant difference[47] | > 0.10 | Treatment success |
| | | > 0.10 | Relapse rate |
| | | > 0.10 | Case fatality rate |
| Ceftiofur and trimethoprim/sulfonamide | Ceftiofur[40] | < 0.05 | Mortality rate |
| | | < 0.05 | Treatment days per case |

[a]Treatment success is defined as the lack of clinical signs of BRD at the conclusion of a predetermined treatment regimen. The converse is death or culling due to the development of a chronic illness.

**Table 2. Antimicrobial resistance in *Pasteurella multocida* and *Mannheimia haemolytica* isolated from cases of bovine respiratory disease, from Toowoomba Veterinary Laboratory, Queensland, Australia (1 January 1997 to 31 August 2000)[a], Iowa State University Diagnostic Laboratory (1 January to 31 December 1995),[50] and Kansas State University Diagnostic Laboratory (1 April to 31 December 1994).[50]**

| | Percentage of P multocida isolates showing antimicrobial resistance | | | Percentage of M haemolytica isolates showing antimicrobial resistance | | |
|---|---|---|---|---|---|---|
| Antimicrobial | Toowoomba (n in brackets) | Iowa (n = 103) | Kansas (n = 54) | Toowoomba (n in brackets) | Iowa (n = 112) | Kansas (n = 60) |
| Streptomycin | 88 (25) | - | - | 50 (16) | - | - |
| Neomycin | 9.4 (32) | 40 | 44 | 0 (22) | 17 | 27 |
| Tetracycline | 3.1 (32) | 14 | 17 | 0 (22) | 24 | 35 |
| Ampicillin | 0 (32) | - | - | 0 (22) | - | - |
| Sulphamethoxazole/ Trimethoprim | 0 (32) | - | - | 0 (22) | - | - |
| Sulphadimethoxine | - | 86 | 80 | 0 (22) | 79 | 88 |
| Lincospectin | 0 (32) | - | - | 0 (22) | - | - |
| Penicillin | 40.6 (32) | 33 | 39 | 0 (22) | 63 | 95 |
| Ceftiofur | 0 (31) | 0 | 7 | 0 (22) | 0 | 3 |
| Tilmicosin | 0 (7) | 9 | 13 | 0 (7) | 6 | 8 |

[a]The Toowoomba Veterinary Laboratory uses the NCCLS Standard Method of Susceptibility Testing using agar disc diffusion. As streptomycin is no longer registered for use in food producing animals in Australia it is no longer tested at the Toowoomba Veterinary Laboratory.

*Anti-inflammatory drugs* - Much of the clinical presentation of BRD is due to respiratory tract inflammation.[34] The severity of the disease can therefore be reduced by the administration of anti-inflammatory drugs.[52]

The use of corticosteroids in the treatment of BRD is contraindicated due to their immunosuppressive effects[51] and their potential to cause recrudescence of BHV1 infections.[53] A dexamethasone dose of 0.04 mg/kg administered once daily is used as an immunosuppresive model in cattle.[54] This is equivalent to only 2.4 mL of 5 mg/mL dexamethasone for a 300 kg animal. Corticosterone acetate treated tissue cultures produced 10 to 12 times more BHV1 than controls,[55] suggesting that corticosteroid treatment or environmental stressors could cause substantial variation in viral excretion from clinical cases of IBR.[18] Supplementation of antibiotic therapy with corticosteroids usually results in poorer responses, increased relapse rate and prolonged illness compared with the administration of antibiotics alone.[56]

Inflammation associated with BRD may be reduced by the administration of NSAIDs. Immune function is not impaired by NSAIDs, and these also have antipyretic and analgesic actions that corticosteroids lack.[56] The anti-pyretic action of NSAIDs may be of particular value in cattle because of the reliance of cattle on respiration for temperature regulation.[57] Flunixin meglumine administered intravenously at 2.2 mg/kg to calves with PI3 induced pneumonia resulted in an improvement in clinical signs and a reduction in lung consolidation.[58] In a study of respiratory disease in young calves, administration of flunixin meglumine concurrently with tilmicosin, compared with administration of the antibiotic alone, resulted in a decrease in relapse rate from 27.9 to 15.5%, although this decrease was not statistically significant ($P > 0.05$).[59] The efficacy of carprofen and flunixin meglumine did not differ significantly ($P = 0.53$) with a single injection of carprofen at 1.4 mg/kg at the time of antibiotic administration and daily administration of flunixin meglumine at 2 mg/kg for 3 days.[60] Flunixin meglumine also has anti-endotoxic activity, which may be advantageous in cases of BRD where *M haemolytica* or *P multocida* play a major role.[51]

Tilmicosin appears to induce apoptosis in pulmonary neutrophils leading to a reduction in leukotriene B4 synthesis, thereby reducing further amplification of the inflammatory injury of BRD.[61]

*Antioxidants* — No reports were found on the use of antioxidants in the treatment of BRD with research concentrating on the potential role of antioxidants in growth and disease prevention.

*Miscellaneous therapeutic agents* — Other therapeutic agents which have been investigated experimentally for the treatment of BRD include bronchodilators, antihistamines, mucolytics, immunomodulators and diuretics.

Bronchodilators such as clenbuterol[51] may be of value in reducing the severity of the clinical signs of BRD by counteracting the decreased tidal volume caused by pulmonary oedema and inflammation and enhancing gaseous exchange in cattle with pulmonary lesions.[62]

Since histamine is not known to be directly involved in the pathogenesis of BRD,[57] the use of antihistamines in the treatment of BRD is likely to do little more than increase treatment cost.[54]

Production of viscous purulent exudate, in response to BRD, often exceeds removal by expectoration, thereby obstructing the airways and impairing gaseous exchange.[51] Mucolytics decrease the viscosity of sputum making it more easily cleared. Bromhexine can improve gaseous exchange in the animal suffering from impaired respiratory function by reducing airway congestion and the accumulation of tenacious mucus.[51] However, a functional mucociliary escalator is required to clear this mucus, which may explain the variable results recorded with treatment of BRD cases with mucolytics. Bromhexine has the added potential benefit of increasing pulmonary concentrations of oxytetracycline, sulfonamides and erythromycin by altering local blood supply and the permeability of the respiratory mucous membranes.[51]

Despite encouraging results using immunomodulators in vitro, in vivo responses have generally been disappointing. Levamisole appears to act primarily on T-cells, perhaps with a secondary action of increasing antibody production by T-cell stimulation of B-cells.[63] The authors of a review of the effects of levamisole on naturally occurring or experimentally induced BRD concluded there was insufficient evidence of benefit to recommend the inclusion of levamisole in feedlot arrival programmes aimed at reducing the incidence or severity of BRD[64]. Interferon has been found to inhibit viral and bacterial replication in vitro, enhance the activity of natural killer cells and phagocytosis by macrophages, inhibit delayed hypersensitivity reactions and regulate the production of antibody.[63] BHV1 was shown to be relatively resistant to interferon inhibition and the authors attributed reduced morbidity and mortality to the immunomodulatory effects of interferon.[65] Due to the short duration of interferon activity (less than 1 week)[65] the greatest reductions in morbidity and mortality due to BRD could be expected in cattle treated with interferon on feedlot entry. However, if mechanisms could be developed to achieve sustained release of interferon over a 6 week period, its value may be greatly enhanced. A parapoxvirus based immunomodulator, baypamun N, has significantly reduced the clinical signs of respiratory disease in calves.[66] Metaphylactic administration of baypamun significantly reduced the mean number of treatment days in calves with BRD, but the prophylactic treatment with baypamun on the basis of regular physical examinations was associated with an increased incidence of BRD.[67]

The use of diuretics is contraindicated in the treatment of BRD because these agents exacerbate dehydration, decrease cardiac output, and increase the viscosity of bronchial secretions.[54]

Further research is warranted on the potential use of bronchodilators, mucolytics and immunomodulators.

## Prevention of bovine respiratory disease

*Vaccination* — Until 2001, vaccines against BRD agents were not commercially available in Australia. This has focused prevention of BRD in Australian feedlots on management factors. Live BHV1 vaccines have not been imported due to the dangers of importing an abortigenic strain; abortion due to BHV1 has not been reported in Australia. Seven trials with a live attenuated Australian strain of BHV1 administered intranasally resulted in a significant improvement in growth rate and feed conversion ratio ($P < 0.05$) without a significant reduction in the percentage of cattle treated for all feedlot diseases ( $P > 0.05$) during the first 30 days on feed (Young, unpublished data, 2000). This indicates the vaccine may have reduced the severity of BRD cases involving BHV1 without reducing the incidence of disease.

Although vaccines against BRD agents have been used in North American feedlots for decades, BRD continues to be a major disease problem in these feedlots.[68] In North America, vaccination has resulted in equivocal changes in the incidence of BRD[68] and many of the studies on vaccination of feeder calves in which adequate control groups were included, suggests the practice does not appreciably reduce the incidence or severity of BRD or have a beneficial effect on growth rate and feed conversion efficiency.[5] Most vaccines in North America against BRD agents have been ineffective in preventing respiratory disease.[69] The explanation for this appears to be twofold. Firstly, a very high proportion of feedlot cattle in North America are purchased through the saleyard system.[70] The absence of a relationship between the feedlot and the property of origin does not provide an incentive for graziers to administer vaccines 2 to 3 weeks before delivery to the feedlot to allow an adequate initial immune response. Administration of vaccines at the time of feedlot entry limits their value in the control of BRD because the majority of cases of BRD occur within the first 4 weeks in the feedlot with cases in the first week being predominant.[32] Secondly, and most importantly, BRD is a complex initiated by one or a combination of stressors that provide a microbiological ecological niche which can be exploited by a number of pathogens. It is, therefore, not feasible to vaccinate against all the potential pathogens that might exploit this niche.

Conversely, Australian feedlots commonly purchase cattle directly from a single property, so appropriate use of vaccines in combination with management practices currently used to minimise stress, has the potential to reduce the incidence of BRD.[13] Further, the efficacy of North American vaccines against BRD agents has been questioned[71] on the grounds that registration has been based on efficacy studies that do not examine naturally occurring disease in commercial feedlots and are not reported in peer reviewed journals.

*Mass medication* — North American studies have illustrated reductions in the incidence of BRD in response to mass medication with injectable antimicrobials. Positive responses to mass medication have been found following administration to all cattle at feedlot entry of benzathine penicillin,[71] long acting oxytetracycline,[72] sulfadimethoxine,[73] and tilmicosin;[74] selective administration on the basis of rectal temperature at feedlot entry of tilmicosin;[75] administration of long acting oxytetracycline to all cattle in a pen once BRD incidence exceeded 5% (no time frame reported);[76] and delayed administration of tilmicosin to all cattle in a pen.[77] In addition to a reduction in BRD morbidity, four of these trials[74-77] also showed a positive growth rate response to treatment.

Meta-analysis has been used to examine the effect of antimicrobial mass medication on morbidity, mortality and growth rate as these related to BRD.[68] Of 107 field trials, only 10 were randomised controlled field trials deemed suitable for meta-analysis. The results indicated that parenteral mass medication with long acting oxytetracycline or tilmicosin on feedlot arrival would significantly reduce BRD morbidity in feedlot cattle. However, the author concluded that data on the effects of mass medication on mortality and performance were unreliable, there were insufficient data on the most effective treatment regimens, and there were no valid data on the efficacy of mass medication delivered in feed or water for prevention of BRD. In a subsequent trial, mass medication with injectable tilmicosin at feedlot arrival was superior to oral chlortetracycline in terms of BRD morbidity and treatment costs.[78]

The outcome of BRD measured in mass medication trials is a complex affected by a large number of factors, some of which are specific to country, production system, region, feedlot or time of year. It may therefore be inappropriate to apply conclusions drawn from North American studies to Australian feedlots. Feedlot specific responses can only be evaluated on individual feedlots and perhaps at specific times of the year.

*Management factors* — An Australian study into the effects of management and vaccination of cattle before delivery to the feedlot[19] examined seven treatments known as 'pre-boosting' that included combinations of vaccination against BVDV, BHV1, PI-3, *P multocida* and *M haemolytica* at least 1 month before feedlot entry; yard weaning with supplementary hay; additional handling and training to eat from a trough; and non-specific immunostimulation (Equistim® at 1 mL/100 kg) given shortly after weaning. An untreated control group was paddock weaned and run with the treated cattle on the research station. Comparisons were made between the treated cattle, the research station controls and commercial in-contact cattle. The commercial in-contact cattle were from a variety of sources including direct from the property of origin and saleyards. There was significantly less morbidity and mortality in the research station cattle compared with the commercial in-contact cattle, with the major disease problem in the commercial cattle being BRD. In comparison with the research station controls, which had better health and performance than the commercial cattle, there were significant benefits from all pre-boosting treatments. Weight gain to day 37 was significantly improved by all treatments (11 to 20%, $P < 0.05$), but total feedlot weight gain was only improved by treatments that included vaccination (8%, $P < 0.02$). During the adaptation phase small benefits due to training procedures were seen but there was no significant benefit over the entire feeding period. Economic analysis showed only three of the treatments were cost-effective. These were vaccination, yard weaning, and yard weaning plus vaccination. Thus, management of weaning alone or in combination with vaccination at least a month before feedlot delivery yielded an economic benefit in reduced disease incidence and increased weight gain during the feedlot phase.

*Manipulation of immunocompetence with antioxidants* — Many trials have addressed the potential effect of increased antioxidant intake on general feedlot health and performance and whereas some have found positive responses,[79] others found no effect on feedlot performance.[80] No reports were found of any trials that have specifically addressed the potential effect of increased antioxidant intake on the incidence and severity of BRD. This is an area which warrants further research.

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# BOOK REVIEW

***Adams' Lameness in Horses.*** *Stashak TS. 5th edn. Lippincott Williams and Wilkins, Broadway, 2002, 1173 pages. Price AUD 242.00. ISBN 0683 07981 6.*

*Adams' Lameness in Horses* has been a landmark publication for the veterinary community and equine industry for over four decades. The first edition was published in 1962 with the predecessor to the latest edition being published in 1987. The intervening 15 years has seen many advances in the understanding of lameness in horses. This ranges from diagnostic imaging to the role of nutrition in musculoskeletal development and disease and the molecular biology of the musculoskeletal system in health and disease. These advances have been very successfully incorporated into the latest edition, which the editor states is "designed to appeal to a wide audience in equine related fields".

There are 17 contributing authors and the chapter structure follows that of the previous edition. It covers functional anatomy, conformation and movement, examination of lameness, diagnostic imaging, role of nutrition in musculoskeletal development, disease of bones and related structures, disease of joint tendons and related structures, lameness, and trimming and shoeing for balance and soundness.

The presentation of the information on shoeing has been rationalised from the last issue, which included four separate chapters on shoeing. However, the total number of pages dedicated to the topic has been increased from 46 to 61. A separate chapter on gaits from the 4th edition has been integrated into chapter 2 'Conformation and Movement'. The chapter dedicated to 'Methods of Therapy' has been deleted, with the editor noting that most of the material is covered when dealing with specific lesions or diseases and that there are many other texts which "cover the topic more completely than I possibly could in one chapter". Extracorporeal shock wave therapy for example is described in sections of the book dealing with bone spavin and tibial stress fractures. However, advances in equine physiotherapy and the use of therapeutic modalities may warrant the reinstatement of a 'Methods of Therapy' chapter in the next edition. The decision to exclude the full description of bandaging techniques such as the Robert Jones bandage from the 5th edition must have been difficult for the editor to make.

The first glance impression by an owner of the 4th edition of this publication, who is considering the purchase of the 5th edition may not be positive. The text of page 1 in both editions is practically identical. The majority of the figures in Chapter 1 are reproduced in both editions. However, there have been modifications to figures such as the one depicting arterial supply to the digit of the forelimb. Advances in the understanding of the functional anatomy such as Pollitt's work on the microcirculation of the equine foot have also been included in the chapter. This is further illustrated in the reference section of the chapter, which has been increased from 27 to 51 citations. The extensive reference sections provided throughout the book are a major attribute for anyone wishing to delve further into particular areas.

Any negative first impressions will quickly disappear as the reader delves deeper into the book. The majority of the remaining chapters have undergone major rewriting, updating and enhancement. The chapter on diagnostic imaging has been expanded to include separate sections on ultrasonography and nuclear medicine and all sections are very well illustrated. However, a comparison of some images that appear in both the 4th and 5th editions indicates that the quality of the reproductions may not be as high.

The 200 page chapter on diseases of joints, tendons, ligaments and related structures is outstanding. State of the art current knowledge and understanding is presented along with a clear vision of what the future holds in the way of diagnosis, prevention and treatment of diseases of these tissues. This is a clear benefit of having world leaders in their fields as authors.

Chapter 8 titled 'Lameness' includes the work of five authors and has 28 sections starting with 'The Foot' and ending with 'The Wobbler Syndrome'. Each of these sections is further divided in up to 30 subsections dealing with specific entities. Information on aetiology, signs, diagnosis, treatment, prognosis and a reference list is presented on the majority of conditions. There is also extensive use of images and figures throughout this section of the book. The definition of some of the nuclear scintigraphy images in this section is suboptimal but it is compensated for in the section of the book dedicated to nuclear medicine.

It is clear that Adams' Lameness in Horses will continue to be a keynote publication for the veterinary and broader equine communities. Additionally if the next edition is to be designed to appeal to a wide audience as well it will need to be presented in two volumes.

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