FOR NSW FEEDLOT MANUAL

PART D – TECHNICAL ISSUES

CHAPTER 14

DISEASE CONTROL IN THE FEEDLOT CATTLE INDUSTRY

CONTENTS

INTRODUCTION

Diagnosis of disease in production systems requires a good understanding of management and feeding regimes. This is particularly true in feedlots, because many of the diseases encountered are related to management and nutrition. Cattle feedlots aim to produce tender, marbled beef by feeding diets with high levels of grain. The rumen is designed to be a fermenting chamber for roughage rather than to handle high levels of carbohydrate. Therefore, the management and nutrition of feedlot cattle must be finely tuned so that digestive and other functions are not out of balance.

INTRODUCTORY HEALTH REQUIREMENTS

Cattle being selected for feedlots need to be healthy, structurally sound and of a suitable frame size to cope with their expected final weight. Cattle entering feedlots often come from many different genetic, nutritional and geographic backgrounds. They may have been purchased in a saleyard, transported long distances and then commingled in the feedlots. Some will be carrying disease organisms and others will have never been exposed to these. The cattle will also experience a marked diet change on entering the feedlot. All of these stresses can lead to disease outbreaks. Research carried out by Dr Lloyd Fell and others of NSW Agriculture[1] has demonstrated that yard weaning of cattle has a beneficial effect on the future performance of feedlot cattle by educating them to eat from a trough. Introductory feedlot animals that are least stressed in terms of health are animals that have been yard-weaned, are older (not immediately weaned) and have been purchased directly from a backgrounding property rather than a saleyard, and have not comingled with strange cattle.

Vaccination

Unless a vaccination certificate accompanies the cattle at entry, the vaccination status is unknown, and clostridial vaccination is recommended. Routinely vaccinate all cattle with 5-in-1 vaccine on arrival and again four weeks later. This vaccine prevents the diseases pulpy kidney, blackleg, black disease, malignant oedema and tetanus.

An infectious bovine rhinotracheitis (IBR) vaccine, Rhinogard®, by Q-Vax, is available in Australia. It is a live vaccine administered intranasally to cattle at the time of entry to the feedlot. Other vaccines that may be required, depending on location and disease incidence, are vaccines against leptospirosis, botulism, anthrax, tick fever, and bovine ephemeral fever.

Other vaccines that are currently being developed for Australian use are pestivirus vaccine and pasteurella vaccine.

Drenches

The type of drench required will depend on the cattle origin and age. Most cattle develop good resistance to roundworms once they reach 18 months to two years of age; drenching older cattle may be uneconomic. Liver fluke can affect growth rates at any age but are regional. Therefore, consider the source of the cattle before treatment. In general, cattle should be drenched for roundworms and liver fluke in the introductory phase of feedlotting. The roundworm drench needs to be effective against immature *Ostertagia* (brown stomach worm).

Tick treatment

This is needed in Queensland only and is done by chemical treatment or vaccine.

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[1] Project DAN.069 – *Reducing Feedlot Costs by Pre-Boosting: a Tool to Improve the Health and Adaptability of Feedlot Cattle (KH Walker, LR Fell, LA Reddacliff and L Davies)*, Final Report 1997

Lice treatment

Use a spray or pour-on lice treatment if lice are present at introduction.

Lice cause some discomfort to cattle. Cattle with lice tend to rub on fences and may damage them. Cattle with lice are also less attractive at saleyards.

Vitamin A

An injection of vitamin A is recommended, particularly if animals have come from dry, drought-affected pasture. Both vitamins and A and E are recommended supplements to feedlot rations. Vitamin ADE injections contain little vitamin E and should not be relied on as the sole supplement of vitamin E.

Vitamin B1 (thiamine)

This is a recommended supplement for the feedlot ration.

Horn tipping

Recommended.

Taking rectal temperatures

May assist in detecting those that are sick at introduction.

DETECTING AND TREATING DISEASE

- Animals must be identified individually. Check feed intake, especially in starter pens. Pen riders must check cattle once or twice per day. They need to be familiar with diseases of feedlot cattle and the signs to expect.
- Sick cattle are removed to a sick pen or hospital pen for further examination and treatment.
- Generally, respiratory disease is most common during the first 25 days.
- It is most important that deaths are investigated. Post-mortem examinations allow identification of diseases that are occurring in the feedlot.
- Each feedlot should instigate a disease control plan in consultation with their veterinarian.

FEEDLOT RECORDS

Records should be kept of treatment at entry and any later treatments, sicknesses (morbidity) and deaths (mortality), and withholding dates following antibiotic treatment. Feedback from abattoirs should be incorporated into the records. A summary of morbidity and mortality rates and disease conditions should be prepared monthly.

FEED-RELATED ILLNESSES

Many of the illnesses experienced by cattle in feedlots relate directly to diet. The high levels of carbohydrate (grain) in feedlot finishing rations contribute to the most commonly observed illnesses. Other problems are associated with high protein diets, excesses of fats or oils, or large proportions of indigestible roughage in the diet. Diet-related problems include:

- lactic acidosis and grain engorgement
- rumenitis, parakeratosis and liver abscesses
- laminitis (founder)

- polioencephalomalacia
- feedlot bloat
- urea poisoning
- indigestion not caused by acidosis
- excess lipids
- urolithiasis (bladder stones)
- vitamin A deficiency
- vitamin E deficiency

Lactic acidosis

Causes

The underlying cause of several feed-related illnesses is lactic acidosis. Ruminant animals are designed to consume large amounts of pasture or roughage. The ruminal fluid in a pasture-fed animal normally has a pH of 6.5 to 7 and a complex mixture of microorganisms (protozoa and bacteria), all capable of breaking down the large amounts of cellulose in roughage. Including large proportions of highly digestible carbohydrates, such as grain, in feedlot cattle rations increases the acidity of the ruminal contents and changes the population of microorganisms inside the rumen in favour of lactic-acid-producing bacteria. If changes to the ration are made gradually, the ruminal microorganisms can change and accommodate the higher acidity. Should the acidity change very quickly or drop below a pH of 5.5, a severe problem can occur: lactic acidosis.

Acidosis may result from:

- a sudden increase in the percentage of grain in the diet
- a rapid change from one grain to another: insufficient time allowed on introductory lower carbohydrate (grain) diets (starter rations).
- feeding finely processed grains
- not enough roughage in the diet
- a sudden change in the diet
- cold weather – cattle often increase their intakes suddenly in cold weather
- not enough buffers such as sodium bicarbonate, sodium bentonite, calcium oxide
- not enough protein.

Clinical Signs

The signs of grain engorgement vary with the severity of the accompanying acidosis. They include: depression and decreased appetite, abdominal pain and depressed or complete stoppage of rumen movements, usually accompanied by diarrhoea. Rumen pH can drop below 6.5 and may drop below 5. All animals usually have soft porridge-like diarrhoea and gut pains; they grind their teeth and kick at their bellies. The speed of onset of clinical signs depends on the amount of grain eaten, the type of grain (wheat, barley and corn are a greater risk than oats or sorghum) and the amount of processing of the grain (increased processing increases the risk).

Treatment

For treatment see Table 14.1

Table 14.1. Treatment of lactic acidosis

Signs	Treatment
Very acute • severely depressed • lying down on its side and unable to stand • may appear blind • body temperature 35.5–38°C • rumen pH below 5 (measure with pH meter or Litmus paper)	May recover if given intensive therapy. Call a veterinarian or destroy animal.
Acute • depressed • unsteady on feet • drinks but doesn't eat • rumen pH between 5 and 6 • body temperature 38.5–39.5°C	Consider emergency slaughter or call a veterinarian. Therapy required includes surgical removal of the rumen contents, plus intravenous fluids.
Subacute • fairly bright and alert • able to walk • may eat and wants to drink • temperature normal 38.5–39°C • rumen pH 5.5–6	Drench with 100 g sodium bicarbonate or 400 g magnesium hydroxide per 450 kg bodyweight. Drench with 500 g magnesium sulfate (Epsom salts) Feed hay. Intravenous fluids if indicated. Animal should eat again within 24–36 hours.

Prevention

Ionophors can help prevent acidosis, and the antibiotic virginiamycin is available from your veterinarian (Eskalin®, Phibro Animal Health) to reduce the lactic acid producing bacteria.

Rumenitis, parakeratosis and liver abscesses

After acidosis, the lining of the paunch (rumen) becomes inflamed (rumenitis). Bacteria may then penetrate the rumen wall, enter the blood stream and proceed to the liver. The liver filters these bacteria from the blood, and abscesses may form in the liver.

Continual low-grade acidosis may also result in rumen parakeratosis. The lining of the rumen thickens and becomes firm and discoloured. It may also peel away.

Animals affected by these diseases often show no symptoms. Both liver abscesses and parakeratosis result in offal rejections at the abattoir. Both are induced by acidosis.

Ration management is the most important preventative measure.

Laminitis (founder)

Cause

The lactic acid produced in the rumen upsets blood circulation throughout the body. This disruption of blood circulation may result in inflammation within the hoof. Lameness is associated with pain caused by inflammation of the sensitive laminae initially and then caused by changes in the structure of the hoof. This must be distinguished from foot abscess.

Clinical signs

Cattle affected with laminitis may display a range of symptoms. In the severely acute form, cattle are reluctant to walk and often lie down. Some prefer to kneel rather than stand, or stand with the front legs crossed. Some appear agitated, moving weight from one limb to the other. In early cases, there may be sweating and muscle tremors. The limb veins may be swollen and the skin near the coronet red. The hoof is usually hot and painful.

Less severely affected cattle tend to walk on their heels, wearing the sole at the heel. The toes elongate ('slipper foot'). The hoof wall over time usually develops horizontal ridges or cracks. These horizontal marks indicate when the acute laminitis previously occurred.

Treatment

Treat immediately if you want to reduce production losses. Seek veterinary help. Treatment of individual animals with antihistamines is warranted if your animals are valuable. Painkillers, anti-inflammatory drugs and the antibiotic, virginiamycin, may all help alleviate laminitis.

On a herd or yard basis, feed hay and remove concentrates from the diet immediately. Reassess the ration you're using before reintroducing cattle to it. Reintroduce the diet slowly.

Polioencephalomalacia (PEM)

The clinical condition known as polioencephalomalacia (PEM), which literally translates as 'grey matter softening', occurs when brain swelling is severe enough to cause death of neurones, particularly in the cerebral cortex because of the increased pressure in the confined cavity of the skull. Young feedlot cattle (six to 19 months of age) are most at risk.

Causes

The condition is commonly associated with thiamine inadequacy (vitamin B 1) or with the presence of thiaminase in the rumen. Cattle normally get their thiamine from that produced by microorganisms within the rumen. It is thought that acidosis encourages microorganisms that produce an anti-thiamine factor, reducing the amount of available thiamine. Several other neurological disorders of toxic or metabolic origin may cause the same lesions – for example, lead poisoning, water intoxication and sulfide poisoning, particularly from ammonium sulfate. Animals affected by these other causes do not respond to thiamine therapy.

Ammonium sulfate is commonly included in feedlot rations as a urinary acidifier to prevent the formation of magnesium ammonium phosphate bladder stones. Ammonium sulfate in the ration has been associated with PEM. Other possible sources of sulfur in the ration include molasses (high in inorganic sulfur), sulfur-containing amino acids (for example, methionine for hoof growth) and drinking water high in sulfur. All dietary sulfur (organic or inorganic) is metabolised to sulfide in the rumen.

Any unbound sulfide is belched up as hydrogen sulfide gas. Much of this gas is in turn inhaled, and at high enough concentrations it is toxic. Several dietary factors may affect the amount of sulfide produced in the rumen and the availability of it for absorption: the amount and form of dietary sulfur; the fibre and fermentable carbohydrate content, which will affect rumen microflora; and the dietary concentration of metals that can affect sulfide bio-

availability. Copper, zinc, iron and molybdenum can form insoluble salts with sulfur, thus decreasing bioavailability. Conversely, low dietary contents of these metals could lead to excessive absorption of sulfide.

PEM has been observed in Australian feedlot cattle at about 20 to 25 days on feed with a ration containing 1.25% ammonium sulfate (with a total sulfur content of 0.5% of the diet). Interestingly, older cattle did not develop PEM after the introduction of ammonium sulfate. This could reflect differences in diet and/or rumen microflora at different stages in the feedlot.

Clinical signs

Affected cattle may die suddenly with no prior symptoms, or may die within 1 to 5 days after developing signs.

Animals affected by PEM display a range of symptoms, including blindness, a dull unresponsive state, a high-stepping gait, stargazing, and occasionally convulsions and collapse. Dopey animals may be ridden by other cattle and identified as ‘bullers’.

Diagnosis

Polioencephalomalacia can be diagnosed at a veterinary laboratory by histopathologically inspecting the brain, but this will not determine the cause. No biochemical test is available to confirm sulfide toxicity, but measurements of total sulfur levels in the diet and drinking water are useful if PEM, unresponsive to thiamine, is suspected.

Treatment

Call your veterinarian. The response to treatment will depend on the cause. If related to thiamine deficiency, initial intravenous thiamine (with repeat treatments) will usually allow an animal to recover entirely if it is treated early.

If the problem is related to sulfur toxicity, some animals will recover if dietary sulfur is reduced.

Prevention

Thiamine in the ration can prevent PEM.

Sulfur-induced PEM can be prevented by reducing the amount of sulfur ingested.

Feedlot bloat

Cattle produce large amounts of gas in the rumen during the digestion of feed. This is normally released up the oesophagus and out the mouth at regular intervals by belching. If the animal can’t release this gas because there is a blockage, or because the oesophagus or rumen is damaged, or because the gas is in a stabilised foam, the gas builds up in the rumen. The swelling rumen will eventually put pressure on the lungs and heart and kill the animal.

Causes

Feedlot bloat is often associated with acidosis. The rumenitis caused by acidosis may stop the rumen functioning correctly, and gas may accumulate. Acidosis may also encourage the growth of slime-producing bacteria that cause foaming of rumen contents, thereby trapping the gas.

Other causes of feedlot bloat include finely processed grain in the ration, low roughage diets, or the type of roughage. Diets with less than 10% roughage, and those containing legume hays (particularly lucerne), may cause bloat.

Clinical signs

Swelling of the abdomen, particularly in the upper left flank. The animal is obviously distressed, standing up or lying down and often kicking at its abdomen. Death may occur very quickly if treatment doesn't start immediately.

Treatment

Stomach tube or drench with a proprietary bloat preparation, vegetable oil (200 mL) or paraffin (200 mL). Stomach tubing is preferred. If the bloat is caused by a blockage rather than froth, relief will be immediate. If it is caused by froth, then the anti-bloat oil is best.

If the animal is lying down and is severely distressed you will need a trochar and cannula or knife. Make a hole 10 to 20 cm long through the upper left flank (paralumbar fossa) and into the rumen. Once the rumen is opened, the gas will be released quickly. Try to keep hold of the edge of the rumen to reduce the amount of rumen contents that spill into the abdomen.

Once the gas has been released, get veterinary help to stitch the wound and treat the animal with antibiotics.

When outbreaks occur, treat individual acute cases. Walk the remaining animals to help foam breakdown and gas release. Watch them closely.

Urea poisoning

Urea is commonly added to feedlot rations as a source of non-protein nitrogen. Poisoning occurs when rations are not thoroughly mixed, or when cattle have access to water containing urea, such as in feed troughs after rain.

Clinical signs

Urea poisoning kills very quickly. In most cases cattle will be dead before you can treat them. Those animals that don't die immediately show signs of severe abdominal pain (teeth grinding, kicking at belly, rolling), muscle tremor, staggering, weakness, bloat and bellowing.

Treatment

Conservative treatment is rarely successful. Drenching with acetic acid or vinegar (5 L every 2 to 3 hours) has been recommended. Removing the rumen contents immediately by surgery is probably the only effective treatment, if it is done early enough.

Indigestion not caused by acidosis

Sudden dietary changes or improperly formulated rations with a high protein content may cause symptoms of indigestion (depression, loss of appetite, slowing of gut movements). In this case, the rumen pH rises (alkaline) because of protein breakdown.

If the rumen pH is alkaline, treatment involves drenching with 5 to 10 L of vinegar and correcting the diet.

Similar symptoms result from feeding a high proportion of indigestible roughage in the diet. Feed intake is reduced and growth rate slows. This indigestion is relieved when you correct the ration.

Excess lipids

High levels of dietary lipid interfere with rumen digestion. The lipids may also bind some minerals and form insoluble soaps. Diets containing greater than 8% lipids present a risk. So if you're adding tallow, as a cheap energy source, don't add more than 8%.

Obstructive urolithiasis

Feedlot cattle are prone to uroliths (bladder stones), which may block the urinary tract. This is because of their concentrate diet and because steers have a narrow urethra, related to castration at an early age.

Causes

Various dietary components will lead to the formation of uroliths with different chemical compositions. Knowledge of the chemical composition will direct the method of treatment and prevention.

Clinical signs

The clinical signs observed in obstructive urolithiasis include signs of abdominal pain (manifest as kicking at the belly), increased respiratory rate, stretching out, treading with the hind feet, grunting, grating of teeth, and occasionally sitting or lying down.

Repeated attempts to urinate can be observed as swishing of the tail, raised tail, twitching of the penis, shaking of the prepuce, abdominal contractions and grunting. White crystalline material may sometimes be observed on the preputial hair. In cases of incomplete obstruction, urine may be seen to dribble out or form a narrow stream, which can often be blood stained. In the case of complete obstruction, rupture of the urethra or bladder occurs in about 48 hours. If the urethra ruptures, urine leaks into the connective tissue of the ventral abdominal wall and prepuce, causing obvious swelling. Because of the irritant nature of urine, the leaked fluid is often blood stained. If the urethra does not rupture, the bladder will greatly distend and may eventually burst.

Apparently, when the bladder ruptures there is an immediate disappearance of discomfort as the urine leaks into the peritoneal cavity. In animals in which the obstruction is not detected early enough and urethral or bladder rupture occurs, the ensuing uraemia causes depression, anorexia, and death approximately two or three days after the rupture.

Post-mortem findings

After death the bladder may be either ruptured or intact. If intact, the bladder is markedly distended and haemorrhagic. The contained urine is often blood stained, and sand-like bladder stones will be visible in the bladder. The physical appearance of the stones varies with the chemical composition. The kidneys may be grossly distended. Peritonitis is a common finding. Aggregates of bladder stones may be found at the point of blockage of the urethra, most commonly at the ‘S bend’ or at the tip of the penis. The urethra before the blockage is inflamed and haemorrhagic, while the urethra after the blockage appears normal.

Rupture of the urethra associated with a blockage results in massive swelling and fluid accumulation in the prepuce.

Laboratory analyses

It is essential that a chemical analysis of the stones be performed to determine the method of treatment.

The specific gravity of the urine can be measured to determine if water consumption is adequate. A high specific gravity may suggest that the animal’s drinking water is unpalatable. Urine pH can be measured with litmus paper or with a hand-held pH meter (available at electrical stores for $100–$200).

Magnesium ammonium phosphate (struvite) uroliths

Factors suspected of contributing to magnesium ammonium phosphate (struvite) uroliths include alkaline urine, sorghum and magnesium.

Alkaline urine. Magnesium ammonium phosphate is insoluble in alkaline (high pH) liquids. Dietary additives can contribute to alkaline urine, for example, sodium bicarbonate and calcium carbonate. Alkaline drinking water can also contribute to struvite uroliths.

Sorghum. Sorghum grain has been implicated in cases of urolithiasis, although the reason for this is uncertain.

Magnesium. High levels of magnesium in the diet and drinking water contribute to struvite uroliths.

Calcium oxalate and calcium carbonate bladder stones

These stones will precipitate in concentrated urine, with calcium oxalate being insoluble over a wide pH range and calcium carbonate insoluble in alkaline urine. Acute and sub-acute acidosis has been associated with increased excretion of calcium in urine. Oxalates are an end-product of catabolism and are normally present in urine. Ingested oxalate can be a problem in animals whose ruminal flora have not adapted to be able to metabolise oxalate.

Treatment and prevention of bladder stones

Surgical treatment is difficult in large feedlot steers. Salvage slaughter at the abattoir or humane slaughter at the feedlot may be the only alternative for steers with complete blockage.

Prevention of all types of bladder stones is aimed at the maintenance of dilute urine, thus preventing the precipitation of crystals.

Drinking water must be of good quality. The addition of salt to the diet encourages drinking. Salt levels can be increased to as high as 4%, but environmental considerations may restrict such high levels of salt usage.

Magnesium ammonium phosphate uroliths. For treatment and prevention, a urinary acidifier can be added to the diet, for example, 1 to 2% ammonium chloride. A cheaper alternative is ammonium sulfate, but there is a risk of inducing polioencephalomalacia.

Levels of alkaline food additives should be reduced. Urine pH should be monitored routinely in feedlots.

Calcium oxalate and calcium carbonate uroliths. Calcium oxalate stones are extremely insoluble over a wide range of pH, so acidifying the urine will not dissolve them (unlike magnesium ammonium phosphate and calcium carbonate stones).

The calcium level must be correct, and the calcium to phosphorus ratio in the diet should be close to 2:1.

Deficiency of vitamins A and E

In feedlots, grain and silage based diets contain inadequate amounts of vitamin E and B-carotene (the precursor of vitamin A) to maintain rapidly growing animals, so supplementation is essential. Deficiencies of Vitamins A and E can occur independently or together.

Vitamin A is necessary for the development of epithelial cells, for normal bone growth, and for the production of the retinal pigment required for adaptation to poor light. Both vitamin A and B-carotene are easily oxidised and are destroyed in the presence of oxygen, heat, light and moisture. Vitamin A (whether stabilised or not) is more rapidly oxidised when trace minerals are mixed with it as a concentrate. The availability of vitamin A from the intestine is affected by excessive nitrates, deficiency of phosphorus, deficiency of vitamin E, infectious disease, parasitic infestations and diarrhoea.

Vitamin A is stored principally in the liver; the level in blood changes little until the liver stores are virtually depleted, and then the levels in the blood fall rapidly. In a feedlot, vitamin A may become critically depleted after 180 days on feed.

Vitamin E is an antioxidant produced by plants and is particularly abundant in seeds in association with their high content of polyunsaturated fatty acids (PUFA). Various forms exist, but alpha-tocopherol is the most important.

Little vitamin E is stored in the body and it can be destroyed or badly absorbed. Alpha-tocopherol acts as a lipid soluble antioxidant, maintaining cell-membrane integrity and permeability. The clinical manifestations of vitamin E deficiency are related to membrane damage, with the organs and tissues affected varying between species.

A combined deficiency of vitamins A and E has been diagnosed in cattle being fattened for the Japanese market when no vitamin supplementation is included in the ration.

Clinical signs

The clinical condition is most obvious in the summer months. Signs include clear discharges from the nose and ears, swollen hind legs, varying degrees of lameness and in coordination, bulging/protruding eyes, glazed bluish appearance to the eyes, poor adaptation to dull light, with pupils more dilated than normal, and occasional cases of complete blindness with no light reflex in the pupils. In advanced cases the swelling or oedema in the hind legs may progress to include oedema of all four legs, and extend to the shoulder and under the jaw. A number of affected animals may have ulcers on their corneas, possibly as a result of injuries incurred through poor vision. Affected animals do not tolerate hot weather and will show increased respiration, drooling and occasionally panting. In the cooler evening, these same animals show no evidence of hyperthermia. Affected animals can die in hot weather and during prolonged transportation.

Laboratory diagnosis

Vitamin A and E levels can be measured in serum or plasma and in tissues, particularly the liver. Samples must be protected from heat and light.

Histopathological examination of tissues, particularly the parotid salivary gland and duct and the eye and optic nerve, will show changes consistent with vitamin A deficiency. Pathological changes to small subcutaneous blood vessels are evident and are most likely related to low levels of vitamin E.

Treatment

Injectable or oral vitamin A will reverse most clinical signs, although occasionally animals will fail to recover.

Prevention

Vitamin A and E requirements are high for rapidly growing animals and may be particularly high when the preference is for marbled meat with a high fat content. The requirement for vitamin A is greater during hot weather, and it is possible that either vitamin A or vitamin E or both are required for effective thermoregulation. Recommendations are given in Table 14.2. The ratio of vitamin A to vitamin E should be between 1000:1 and 1000:5. The proportion of vitamin A could be lower than this, but not higher.

Table 14.2. Vitamin A and E recommendations for feedlot cattle (per beast per day)

Type of animal	Weight (kg)	Vitamin A (IU)	Vitamin E (IU)
Calves finishing as	180–450	30,000–50,000	150–200

short yearlings			
Finishing yearlings	270–500	50,000	150–200
Finishing 2 year olds	360–550	50,000	200–500

Prevention

The recommended levels of supplementary vitamin E for feedlot cattle have changed considerably over the last decade with the increased understanding of the role of vitamin E in enhancing the shelf life of meat, and its role in alleviating stress-related conditions and enhancing the immune system.

In 1982, the recommended level was 20 to 50 IU of supplemental vitamin E per head daily during the finishing period. In 1994, American researchers recommended levels of 500 IU per head daily for the last 100 days of feeding for enhancing meat preservation. American and Japanese researchers have recommended even higher levels of 1200 to 1300 IU per head daily for the last 38 to 67 days of feeding.

The recommended levels of supplementary vitamin E are given in Table 14.2. It may also be necessary to routinely measure the actual levels in the pre-mix as supplied to the cattle. Vitamin E can be quickly depleted in the presence of minerals and alkaline pH, and it is not uncommon for the vitamins, minerals and limestone to be premixed and stored.

PREVENTING FEED-RELATED ILLNESSES

Care with ration formulation and skilful cattle management will minimise feed-related illnesses.

Observe all cattle in each pen at least once daily for early signs of illness. Check feed troughs as an indication of appetite. Move slowly through each pen looking for evidence of bloat, scouring, animals standing away from the mob or hunched up, dehydrated or acutely lame. Autopsy all dead animals. Record abnormal findings in a notebook (date, pen number, number of cattle affected, symptoms) to allow closer study of disease patterns. Computer recording systems are now available to help you.

Introduce new rations to cattle gradually. Cattle straight from the paddock take up to four weeks to adjust to a full feedlot ration. The compromise with economics is to allow a minimum of 14 days to change from mainly roughage to mainly grain diets.

Change ration components gradually, mixing the new component with the old in increasing amounts over at least 10 days. This applies particularly to changes from one type of grain to another.

Maintain regular feed intake by generally not allowing troughs to empty completely between feeding. However, at least two or three times a week, clean the troughs out completely to ensure no old or mouldy feed accumulates in the bottom of the trough. This also ensures that additives such as urea cannot settle to the bottom of the trough and accumulate into lethal concentrations.

Alternatively, you may decide to allow cattle to empty the trough between feeds. If you do this, you will need greater trough space, compared with having feed available nearly all the time.

Cold, wet windy weather increases the appetite, so increase the roughage content of the ration and feed more often to avoid acidosis and spoilage.

Avoid overmilling grain and roughage. Rolled or steam flaked grain mixed with coarsely chopped roughage helps reduce acidosis and bloating. Watch for bloating if lucerne or clover bay is included in the ration.

Build feed troughs with sloping floors to allow drainage after rain. Remove any build-up of old feed from troughs before it goes mouldy.

Consider ration additives such as buffers, ionophores and antibiotics, especially during ration build-up or during prolonged bad weather. Additives must be well mixed into the ration.

Ration additives

Each enterprise must assess the usefulness of any product after considering the ration, previous experience of disease and cost.

Buffers

Sodium bicarbonate (2%) plus sodium bentonite (2% dry matter) improve daily ration intake and daily weight gain and reduce acidosis in cattle being introduced to grain rations. They usually provide little ongoing advantage beyond the introductory period. Adding sodium bentonite at up to 4 to 5% of the final ration may help avoid acidosis if you expect a sudden increase in grain consumption (for example, in cattle on finishing diets in bad weather).

Ionophores

Modifying the rumen fermentation by adding ionophores (including monensin, lasalocid and salinomycin) results in improved feed conversion efficiency with both high and low roughage rations. More energy is made available for weight gain from a given intake. An overall improvement of 7 to 10% in feed conversion is common in cattle on high grain diets, with daily feed intake reduced by 4 to 5%.

The ionophores inhibit several organisms responsible for fermenting carbohydrates to lactic acid, reducing the risk of acidosis on high grain diets.

Note that these can be toxic and cause death from heart failure. This occurs when the ionophor is not well mixed in the feed or when a ruminal capsule breaks open in the rumen.

Virginiamycin

Eskalin® is available as a feed additive to reduce levels of lactic-acid- producing bacteria and hence the likelihood of lactic acidosis.

Antibiotics

Low-level feeding of antibiotics can greatly reduce the incidence of liver abscesses and may improve growth rates. Using broad- spectrum antibiotics in the feed (such as tetracyclines) tends to be reserved for treating specific diseases. Antibiotics must be used as directed by a veterinary surgeon, and you must follow the withholding periods on the label. There is a growing trend away from the use of antibiotics in feed because of the expected development of antibiotic resistance and the real threat this is to human and animal health.

HEAT STRESS

There have been a number of serious episodes of deaths in feedlot cattle related to heat stress, although, in general, heat stress in lot-fed cattle rarely causes death, but can cause production loss.

Cattle get stressed when their body temperature rises because of environmental factors like solar radiation and air temperature, and because of metabolic heat production from diet, exertion and fat insulation. Heat stress causes physiological change.

The secret of managing heat stress is to observe the early behavioural signs and take simple and cost- effective remedial action before the situation gets too severe. Failure to act early can mean that animals will suffer severe stress and you will lose production.

In a publication by Young[2], behavioural signs were listed. These can be used as a reference guide to help you define physical levels of heat:

Signs of heat stress

1. body alignment with solar radiation
2. shade seeking
3. refusal to lie down
4. reduced food intake
5. crowding over water trough
6. body splashing at water trough
7. agitation and restlessness
8. reduced or stopped rumination
9. grouping to seek shade from other animals
10. open- mouthed and laboured breathing
11. excessive salivation
12. ataxia/inability to move
13. collapse, convulsion, coma
14. physiological failure and death.

Animals can usually cope well until sign 9. The onset of 10 is a clear signal that an animal is failing. With shallow breathing, an animal can effectively evaporate moisture from its upper respiratory tract, but with open- mouthed breathing evaporative capacity falls dramatically. Most heat is lost through the skin, either by sweating or by conduction. Hot blood from the body core is brought to the skin surface by small vessels in the skin called arteriovenous anastomoses (AVAs), which connect arteries directly to veins. When needed, they open up, and blood bypasses the slow capillary network, allowing a more rapid flow of blood to the surface, and quicker dissipation of heat from the superficial veins. This mechanism is most important in the distal limbs;, hence the common observation that hot or fevered animals will stand in dams.

Treatment and prevention

- expose cattle to increased air movement
- provide cooled drinking water
- move animals to a shaded area
- apply water sprays

[2]Implications of excessive heat load to the welfare of cattle in feedlots. *Recent Advances in Animal Nutrition* 1993, p. 45

- change the feed type and/or level
- minimise physical exertion
- release animals from close confinement.

Some of these solutions are more cost effective and practical than others. Other preventive measures include using heat- resistant breeds and monitoring weather conditions to provide early warning.

Coat type and colour also influences the susceptibility of cattle to heat stress. White reflects more, and absorbs less incident radiation than does black. Hence, provided the skin is pigmented, a white coat should be better than a black one for cattle in tropical areas; brown and red are intermediate.

Shade has often been promoted as the simple solution, but recent research has shown that shade is only part of the answer. Shade can be a critical factor on certain days in certain situations, but it doesn't eliminate heat stress because humidity, air movement and the type of cattle are such important factors.

Vitamin E depletion and heat stress in feedlot cattle

Research at the Regional Veterinary Laboratory in Armidale[3] has shown that depletion of vitamin E in feedlot cattle is responsible for damage to small blood vessels in the limbs, particularly in the lower hind legs. If this damage progresses it can impair the body's temperature regulation and the animal cannot be able to cope with heat stress.

Leg swelling was detected in three feedlots before deaths from heat stress. Nutritional supplementation with vitamin E was absent or inadequate at these feedlots.

In very hot climatic conditions it seems likely that subcutaneous limb vessels damaged by a lack of vitamin E are unable to perform their function in heat regulation. The resultant rise in body temperature may lead to irretrievable tissue damage.

Clinical signs

The condition is first observed as lameness, with fluid swelling of the hock joint progressing to generalised swelling down to the hoofs and sometimes up to the groin. The skin directly above the hoof and around the pastern is reddened, swollen and tight.

Affected animals may have elevated temperatures.

Animals on feed for less than 60 days are apparently not at risk.

Nutritional factors

The condition occurs in animals fed low levels of supplementary vitamin E. Natural vitamin E in stored feed in silos is destroyed quickly, and there is an apparent increased demand for vitamin E during hot weather. Secondary dietary factors thatwhich may make the condition worse are high levels of fat (tallow or whole cotton seed), high levels of carbohydrate and lactic acidosis, rancidity, fungal contamination, nitrites, and ionophors (for examplee.g. rumensin).

Climatic conditions

The condition is linked to prolonged hot weather, and affected animals improve after the onset of cooler weather.

[3] Vanselow BA (1997) Vascular damage and heat stress related to low antioxidant levels in feedlot related rations. *Recent Advances in Animal Nutrition in Australia* p. 239

Laboratory examinations

Microscopic examination of subcutaneous blood vessels of the limbs reveals pathological changes. Serum or plasma vitamin E levels are low or marginal.

ILLNESSES CAUSED BY FEED CONTAMINATION

- botulism
- ergotism
- mycotoxicoses
- chemical residues

Botulism

Cause

Botulism is caused by a toxin produced by the bacterium *Clostridium botulinum*. The botulism organism multiplies in decaying animal or plant material that has previously been contaminated by that organism or its resistant spores.

Poultry litter, poorly made silage and spoilt hay, brewers' grain and grain with a high moisture content have all been toxic. Chicken carcasses in poultry litter have been a potent source of toxin. Removal of the carcasses by sieving doesn't make the litter safe, as the organism may have been present in dung from the birds and the toxin may have been released from decomposing carcasses. Poultry litter should be kept dry and heat treated to destroy the toxin before it is included in rations.

Note: While properly treated poultry litter is a perfectly safe source of required nutrients, 'chicken litter' or 'dehydrated chicken litter' is excluded as a recommended feed ingredient and has been prohibited for use as such (in feedlot rations) by members of the Australian Lot Feeders' Association. Therefore its use is unacceptable by any feedlot accredited under the National Feedlot Accreditation Scheme.

Clinical signs

Signs usually appear 2 to 16 days after animals eat the toxic material. A progressive muscle weakness leading to paralysis is characteristic. Initially animals are uncoordinated and are later unable to get up. The tongue may be paralysed and saliva may drool from the mouth. As the chest muscles become paralysed, the beast heaves to breathe. Death follows a few hours to a few days after the start of the signs. Most affected animals die, and response to treatment is poor.

Finding the toxin in feed is difficult, as signs occur some days after feeding and the toxin may be present only in pockets throughout the feed. The final diagnosis often depends on eliminating other causes of disease.

If any feed component is suspected of carrying botulism stock can be vaccinated against the disease. Discuss this with a veterinarian. (Note that although botulism is a clostridial disease, 5-in-1 vaccines don't protect against it.)

Keep botulism in mind when you're deciding whether to use feed that is water damaged or mouldy fodder and by-product feeds. Check your livestock insurance to clarify whether botulism is included or specifically excluded.

Ergotism

Cause

Grain contaminated with a specific fungus may cause nervous signs, gangrene of the extremities, or hyperthermia. The fungus (ergot of rye) usually infects cereal rye and, less commonly, maize and ryegrass during warm, wet seasons. Cereal crops infested with ryegrass and harvested for stockfeed following a wet spring may produce ergotism when fed to cattle.

Clinical signs

The acute form of ergotism is rarely seen, but involves convulsions leading to death. Feedlot cattle with ergotism are more likely to show gangrene of the extremities, particularly the lower parts of the hind limbs, tail and the ears. A reduced feed intake, slow weight gain, failure to shed the winter coat and scouring may also occur.

Recently, a syndrome of high body temperatures (hyperthermia), panting and salivation has been attributed to ergotism.

Treatment and control

Remove tile affected grain from the ration and seek veterinary help in treatment of gangrene. The dark, often misshapen, ergot-affected seeds are readily seen in contaminated grain.

Mycotoxicoses

Mycotoxins are toxins produced by fungi growing on feedstuffs. The aflatoxins are the best known. Acute mycotoxicoses are relatively rare, but the chronic form can reduce productivity and impair the animal's immunity to infections. Cattle appear less susceptible to mycotoxins than other livestock. Ammoniation of feedstuffs has been shown to destroy the mycotoxins.

Chemical residues

Some hormone growth promotants and all drenches, dips and antibiotics used in feedlots have withholding periods. Consult the label on any product for the withholding period before using it on cattle destined for slaughter. Also check for your particular export market destination whether and what particular minimum residue levels (MRLs) apply for products you use.

Withholding periods nominated on the label (as required by Australian law) may not be sufficient to meet export requirements in some markets.

If you treat individual animals with any product with a withholding period you should identify them. Further, if the rest of that pen is likely to be slaughtered before the end of the withholding period, the treated animals should be segregated.

Residues of chemicals, unintentionally applied to cattle, may also occur. Examples include contaminated cottonseed products and other by-products from the cotton industry, or if cattle eat grain from silos treated with organochlorine pesticides or get hold of materials treated for control of termites. Most of these 'hot spots' have been identified and rectified, but you should still be assured by your suppliers that their grain doesn't contain pesticides or herbicides. Iif you need to use any pesticide or herbicide in or around the feedlot, take care to use it for only its registered purpose, and avoid exposure of cattle and/or feed sources where indicated.

MAIN INFECTIOUS DISEASES

This is not intended as a complete list of infectious diseases that occur in feedlot cattle. A textbook on cattle diseases is recommended.

- bovine respiratory disease (BRD)

- pinkeye (infectious keratoconjunctivitis)
- foot conditions, including foot abscess
- the clostridial diseases: enterotoxaemia, blackleg, malignant oedema, tetanus
- bovine ephemeral fever

Bovine respiratory disease (BRD)

Bovine respiratory disease (BRD) is the most common cause of death and illness in Australian feedlots. With the changing trends in feedlot management in Australia (bigger feedlots, younger cattle and longer transport distances), it is likely that respiratory diseases will assume even greater importance. In the USA more than 50% of deaths in feedlots are caused by respiratory disease complicated by pneumonia.

Causes

Several viruses and bacteria cause respiratory tract disease in cattle. These organisms are present in Australian cattle populations. The severity of the disease not only varies with the type of organism, but also depends on many other factors. Infectious bovine rhinotracheitis (IBR) virus and the bacteria, *Mannheimia haemolytica* (previously *Pastuerella haemolytica*) and *Pastuerella multocida*, are commonly involved.

Transport, handling, temporary deprivation of feed and water, mixing with cattle from different properties, inclement weather, confinement in unfamiliar surroundings and previous exposure to the organisms all affect the occurrence of respiratory disease. In the USA these factors combine possibly with a virus and allow *Pasteurella* spp. to produce the syndrome called shipping fever. Within 3 weeks of entry to the feedlot, up to 50% of cattle may be affected and about 5% of these die.

Clinical signs

Signs of respiratory disease may vary from a mild, barely detectable illness lasting a few days, with discharge from nose and eyes, fever, coughing and reduction in weight gain, to a severe, often fatal pneumonia, with animals off feed, reluctant to move, standing apart from the mob and showing difficulty in breathing. Some cattle may be found dead with no previously detected signs of respiratory illness.

Treatment

Treatment varies with the disease. No treatment is necessary or suitable in cases of viral infection of the upper air passages. Antibiotics, either by injection for individual animals, or in the feed or water to treat a pen, are often used to treat bacterial pneumonia, or may be given in cases of viral infection to prevent complications. The major problem with medicated feed or water is that they are consumed least by the sick animals that need them most.

Antibiotics must only be used only as prescribed by a veterinarian, who will assess the severity and type of disease present and the likely response to treatment before deciding on the preferred antibiotic and method of administration. Observe withholding periods after antibiotic therapy. You will need to identify animals fully and keep proper records of treatment to make this easy.

Control

Minimise stress on feedlot cattle to reduce respiratory disease. If possible, avoid mixing batches of cattle from different origins until after they have acclimatised to the feedlot. If bovine respiratory disease is a persistent problem in your feedlot, consider taking the temperature of all cattle at induction and possibly at any subsequent processing.

Treat any animals that have a high temperature. Discuss this with your veterinarian. Keep handling on arrival to a minimum. Where possible, buy cattle direct from farms to avoid the extra stresses and time off feed that they suffer at saleyards. Discuss with your veterinarian the medication of newly introduced cattle. Bear in mind that such medication may mask the disease to the extent that the number of severe cases increases.

An IBR vaccine is now available in Australia. It is a live vaccine, administered intranasally and given as one dose to cattle upon entry into the feedlot (Rhinogard® by Q-Vax). At present there are no other commercially available vaccines in Australia against this disease.

Pinkeye (infectious keratoconjunctivitis)

Cause

Pinkeye is a very contagious disease, being readily spread by flies or dust. The bacterium *Moraxella bovis* is commonly isolated from pinkeye cases. There are other causes of keratoconjunctivitis, but they do not cause disease as often as *Moraxella bovis*.

Clinical signs

The steps of infection are:

- watery discharge from the eye
- conjunctivitis
- white or cloudy cornea, possibly leading to perforation
- ulcer on the cornea
- pus inside the eye.

Treatment

- Isolate affected animals, preferably in a darkened area.
- Treat early with an antibiotic ointment from a veterinarian.
- Don’t use a powder for treating pinkeye. Powders don’t maintain adequate levels of antibiotics in the eye and are irritant.
- Severe cases may need injections of antibiotics and antiinflammatory drugs around the eye; the eyelids may need to be stitched together.
- Patches that are glued around the eye to cover it may be useful.

Foot conditions, including foot abscess

Lameness is common in feedlot cattle and compromises feeding efficiency in affected animals.

Causes

- traumatic injury: nail/stone injury, heel bruising or hoof overwear or overgrowth
- laminitis: signs of feed-related lactic acidosis with lameness related to systemic changes; no local feet lesions are visible; usually more than one foot is affected
- foot abscess (‘footrot’) – signs of severe lameness, usually in one foot, fever, immobility and loss of appetite; the foot is hot, painful and swollen and has a cleft or fissure, usually in the interdigital cleft or heel area and sometimes the coronary band area.

Treatment

This must be done early on the day the animal is first seen lame for best results. Antibiotic injections in the hospital pen are essential.

Prevention

- Avoid chronically muddied yards by good design, drainage and pen surface management.
- Remove and treat early cases immediately to contain possible spread. Stones, sharp gravel and rough concrete aggregate should be removed, repaired or covered in yards, laneways and pens.
- Bathing the foot in a copper sulfphate and/or formalin solution may help quickly to reduce affected case numbers if all other measures are in place.

Clostridial diseases

Causes

Clostridia are anaerobic bacteria, i.e. they live without oxygen and to most species of clostridia, oxygen is toxic. Their normal habitat is soil and the intestinal tract of animals and man. They have the ability to form spores that give them the ability to survive in hostile oxygen-rich environments such as soil. Spores can survive in soil for years and are resistant to disinfection and heat. They can also remain dormant in animal tissue such as muscle. The spores germinate when conditions are suitable i.e. low oxygen levels as occurs in bruised tissue.

Clostridia produce enzymes such as proteinases (including collagenase which breaks down collagen, the basic framework of all organs), deoxyribonuclease (breaks down DNA), lecithinase (breaks down lecithin which is a major component of nerves). Some clostridia also produce toxins such as the neurotoxins of tetanus and botulism, epsilon toxin of enterotoxaemia and haemolysins which break down red blood cells. Proliferating clostridia produce large amounts of gas, particularly carbon dioxide and hydrogen from the fermentation of sugars.

Clostridia can proliferate in the gastrointestinal tract when there is a sudden dietary change such as an increase in starch content.

The clostridial species involved in cattle disease:

1. *Clostridium botulinum* causes Botulism (see previous section)
2. *Clostridium tetani* causes Tetanus
3. *Clostridium chauvoei* causes Blackleg
4. *Clostridium novyi* causes Black Disease (often in association with immature liver fluke invasion)
5. *Clostridium septicum, chauvoei, perfringens, novyi* cause Malignant Oedema
6. *Clostridium perfringens* causes Enterotoxaemia, also known as Pulpy Kidney

Clinical signs

The signs are different for each clostridial species. Tetanus causes muscular spasm and can have a high mortality rate in young cattle. Blackleg causes localised swelling in affected muscles and gas can usually be felt under the skin. It often occurs in an area of bruising from poor handling or cattle fighting. The animal has a fever and usually dies in 12- 36 hours. Black disease is a clostridial lesion in the liver and occurs following damage to liver tissue by

things such as liver fluke or tapeworm cysts. Malignant oedema is an acute wound infection causing a localised hot, painful, gassy swelling with fever and profound systemic toxaemia resulting in death. Pulpy kidney is an acute toxaemia, caused by the proliferation of *Cl. perfringens* type D in the intestines and the liberation of toxins often associated with high starch diets. Clinically the animal can have diarrhoea, convulsions, paralysis and a rapid death. Quite often animals are found dead having had no obvious clinical signs.

Post-mortem signs

Blackleg

Incision of the affected muscle shows dark, swollen tissue with a rancid smell and bubbling gas. All skeletal muscle and cardiac muscle should be examined. Bloating and putrefaction occur quickly and blood stained froth can be seen coming from the nostrils and anus. Care must be taken not to misinterpret putrefaction alone as a clostridial disease condition. Large feedlot cattle can putrefy quickly particularly in hot weather. Clostridia are a common post-mortem invader. Keep records as accurate as possible on the time of death and the time period before the post-mortem examination as well as the ambient temperature.

Black Disease

A similar lesion to above but localised in the liver.

Malignant oedema

A similar lesion to above but localised to wound site.

Treatment

Clostridial diseases tend to be rapidly fatal, but clostridia are sensitive to penicillin and treatment may be successful if treatment is early enough.

Prevention

5-in-1 vaccine will help prevent tetanus, blackleg, black disease, malignant oedema and enterotoxaemia. There is a separate vaccine for botulism

Bovine ephemeral fever

Bovine ephemeral fever has the potential to devastate feedlot cattle and has been reported in Queensland particularly in small feedlots. The disease occurs in the summer each year or every second year, predominantly in coastal regions of Queensland and NSW. Occasionally the disease spreads inland under suitable climatic conditions. In large feedlots the insect vectors appear to only inhabit the periphery of the feedlot and affect cattle in the outer pens.

Cause

Ephemeral fever is a viral disease transmitted by the blood-sucking insects: sandflies and mosquitoes.

Clinical signs

Large animals are the worst affected. Clinically there is a fever which may have morning remissions, lameness, recumbency and nasal and ocular discharges. If animals have access to water the disease is not usually fatal except in cases of persistent recumbency.

Treatment

Anti-inflammatory drugs such as phenylbutazone or flunixin meglumine cause remission of signs. Proper nursing of recumbent animals is required.

Prevention

If a real threat exists in a region, preventive vaccination may be necessary. Two vaccinations at least two weeks apart are required for effective protection before the disease enters the area.

RULES AND REGULATIONS

You should be aware that several uncommon diseases are notifiable. These are:

- anaplasmosis
- anthrax
- babesiosis
- cattle tick
- enzootic bovine leucosis
- Johne's disease
- trichomoniasis
- tuberculosis

If your cattle have these diseases or certain chemical residues they may have to be quarantined. Feedlots are allowed to take cattle from Johne's disease positive properties, but cattle from the feedlot can only be sold only to abattoirs that accept Johne's-positive cattle. (Contact NSW Agriculture for a list of abattoirs).

Get veterinary help to detect and diagnose disease early. Diseases exotic to Australia must always be considered in disease investigations. Thorough records relating to the origins of stock in the feedlot are useful when animals are traced from infected properties to the feedlot.

If you use any chemical, drug or pesticide on cattle, or in stored fodder, in buildings or surrounds, you must use it only according to its registered use as described on the label, or as directed by your veterinarian. You must observe the withholding periods for all drugs. If you are using drugs that have a withholding period you should keep accurate records of treatment. Also, unless treated animals are segregated, they should be clearly identified in a distinctive manner. For example, you can use an extra ear tag of a colour kept exclusively for cattle treated with a substance that requires a withholding period.

Drugs in Schedule 4 (S4) under the Poisons and Therapeutic Goods Act 1996 (for example, antibiotics) can be obtained only on prescription and must be used as directed by a veterinarian. Negotiate a day-to-day working relationship with a private and/or consultant veterinarian to meet the many and varied animal health requirements of your feedlot before, during and after it is established.