Chapter 6

Animal health and production issues

The Department of Agriculture contributed to the animal industries through the development and/or introduction of new management approaches and identification of the causes, development of control measures, and in some cases eradication of major diseases. Much of this work was outstanding in national and international terms and provided the firm foundation for Western Australia's livestock industries.

This chapter lists issues covering specific work on animal health problems caused by infection, nutrition or management and two industry-related issues. A major component of the work on management in the livestock industries does not lend itself readily to that treatment. The first pages deal with these issues under the heading of Animal Production.

Animal production

Production research really began with the focus on fat lamb production in the early 1930s, and expanded in the post-war period, shifting emphasis depending on the issues of the time. Similar work was carried out for the southern beef industry after 1960 as staff resources permitted. While research relevant to the sheep industry was also carried out at the University of WA and CSIRO, particularly after World War II, the Department of Agriculture was the only organisation servicing the cattle and other industries at farm level in WA.

Sheep and wool

Fat lambs
In the 1930s the focus was on developing both the fat lamb industry and legume-based pastures.

There was a need to demonstrate the logic of the use of crossbred ewes (usually Border Leicester rams over Merino ewes) with terminal sires (basically Dorset and Southdown). This work was done mainly at Avondale. It was very important as farmers in the medium rainfall areas turned to fat lambs as an alternative to cereals following the 1929 Depression. These farmers would have had little or no knowledge of the breeds and husbandry needed to produce high quality fat lambs. The work included general supplementary feeding and focused feeding (flushing) of ewes before mating. It was shown that feeding a grain supplement to ewes in the last four to six weeks of pregnancy improved the survival of the ewe and lamb.

In the post-war period the increase in wool prices shifted the economics of fat lamb production towards the use of Merino ewes as opposed to crossbred ewes. In later work it was shown that a high protein diet before and during mating increased ovulation and lambing percentage (see below under sheep fertility). At that time there was also some detailed work on the exact mechanism involved in this stimulation of ovulation. This effect appeared to favour later mating and spring lambing when feed quality was higher. However, as high protein lupin stubbles became available from phomopsis-resistant crops, this may not necessarily have been the case.

Post-war research
During the post-war period the general thrusts of the research and extension activities of the Division of Animal Production were in continued work on sheep nutrition and reproduction; the effects of worms on productivity; management of flystrike; time of
shearing and lambing; stocking rates; objective measurement of wool; objective selection for desirable characteristics in the breeding industry; use of hormones to increase fertility in ewes and growth rates in wethers; interaction between pastures and the grazing animal; selection for fleece rot resistance; prevention of acidosis or grain poisoning; the problem of dust and the behaviour and management of sheep in export feedlots and during shipping (see below); the impact of nutrition on wool growth in autumn; and the value of ammonia-treated grain to increase protein content.

A computer model was developed to help vital decisions such as how many sheep to run in a paddock, and how much phosphate fertiliser to apply. The model looked at prices and costs and calculated the point of optimum return based on wool production as influenced by the effects of fertiliser rate and stocking rate on pasture production.

Management for survival of lambs immediately after birth and of weaners through the first summer/autumn was always critical. It was shown that the body weight of ewes at mid-pregnancy was an important factor. This was important, as work showed that as many as one in five Merino lambs died in WA between lambing and weaning.

During the rapid expansion of cleared area and area of sown pasture in the late 1950s and the 1960s there was an acute shortage of sheep. There was a dramatic fall in sheep numbers after the collapse of wool prices in 1990, and a shortage of sheep returned from around 2006 and became more acute as the prices paid for wool and lambs increased. In the 1960s work to increase the number of lambs produced per ewe included using rams from the high fecundity Booroola strain. However this demonstrated the need to ensure that the lambs survived through the early days after birth. In general, the more intensive management needed to ensure a high survival rate has not been available on the large farms in the State.

In the 1960s housing was also tested. While it improved survival, it was not practical. Overall this work showed the importance of close management and good nutrition during the lambing cycle.

At all times of lambing, weaner nutrition is critical to maintain growth rates (and survival) through the first summer/autumn. A lot of work was carried out over the years using different techniques for preserving feed quality or different direct supplements. The use of early-mown pastures was one technique tested as a mating supplement and feed supplement for weaners. The use of phomopsis-resistant lupin stubbles was also tested and this higher protein feed was shown to be an excellent diet for weaners.

It was calculated that if half of the seven million weaners in farming areas were grazed on lupin stubble this would yield an extra $15 million for the industry. Good nutrition to achieve high growth rates was particularly important if the mating of weaner ewes was planned. On the domestic market lambs carried over for the high-priced late summer to early winter markets need special attention. Considerable work was done on lamb nutrition, including lot-feeding. The effect of weaning of lambs several weeks before slaughter on carcase weight and quality was examined.

**Lambing times and nutrition**

The industry has traditionally mated ewes in the early summer for late autumn lambing. In the State’s Mediterranean environment, with reliance on annual pastures, late autumn is often a time of limited feed supply and quality. For this reason the department recommended that the industry shift lambing from autumn to spring when feed supplies for the milking ewe and growing lamb are normally available and increasing. The increased feed supplies in early spring reduced the level of supplementary feeding required in late pregnancy and reduced the potential for pregnancy toxaemia in the ewe. In earlier times this did not suit the export fat lamb industry which aimed at a window of
opportunity from September to November before the UK market was supplied by New Zealand. After the UK entered the Common Market in 1971 this market was largely lost. While the logic seemed right the adoption of spring lambing by the industry was limited.

**Objective measurement in breeding**

Work based in NSW showed that improvement in the genetic capacity of a breed could be best achieved by selection of breeding stock on the basis of measurement of key characteristics. In a wool industry focused on fleece weight and the micron thickness, measurement was clearly demonstrated as being superior to the traditional visual assessment used in the Merino stud industry. The department promoted this technology heavily to the industry, with limited initial success. However, over time the industry moved to objective measurement in its breeding programs.

**Stocking rates**

Following the demonstration by CSIRO at Glen Lossie Research Station that higher stocking rates were both possible and desirable, a lot of testing and demonstration of this change was carried out across the State. A report of work on the interaction between fertiliser and stocking rates is in Chapter 7. These demonstrations were important in lifting stocking rate in the industry and improving profitability. This work also demonstrated the importance of testing pasture species under grazing, resulting in the productivity of some different pasture species being tested. It showed, among other things, that a species which is showy in spring but has a low seed set and a sparse autumn germination did not do well under intensive grazing. Another example was the performance of medics of similar visual appearance in a large-scale grazing project on a red clay loam. The new medic species being tested proved capable of carrying 60 per cent more sheep without penalties in wool or liveweight production than the established (control) medic Cyprus.

**Wool: ‘Sale by sample’**

Historically, the assessment of fleece quality by buyers and producers was visual. CSIRO developed equipment capable of assessing the fineness of the fibres in microns. Starting in the early 1970s fleece measurement became the standard practice in the industry and wool was sold on its clean wool weight and the micron measure. Initially instruments measured the average micron of the wool sample. Subsequently instrumentation to measure the distribution of the fibre thickness in a sample was developed. This character was shown by the department to be heritable. The department was heavily involved in promoting this change in a conservative industry.

**Time of shearing and wool quality**

In WA ‘tender’ wool had been a continuing quality problem, with 36 per cent of wool offered at some sales affected. This resulted from the practice of spring shearing so that the weaker, summer/autumn wool was in the middle of the fibre strand. In order to address this problem the department recommended and promoted autumn shearing, which resulted in the weak growth being at the ends of the fibre.

The uptake by the industry was variable and there remained a problem for spinners in dealing with the weaker ends on the autumn-sheared fibre. The CSIRO wool research laboratory showed that tender wool could be processed as well as other wools if the settings on the card were set appropriately. In recent years, the department, in association with spinners in Europe, has demonstrated this technology commercially. It was proved that with adjustment to machines both types of wool could be spun into an excellent product. Nevertheless there remained a strong case for better feeding of sheep through the summer/autumn. In 1993 the Department of Agriculture joined the Cooperative Research Centre for Premium Quality Wool established to look for ways to strengthen wool fibre.
The live sheep trade
The live sheep trade has been an important and growing part of the market for the past 50 years. Initially the shippers sought heavy older wethers. A study tour by a departmental officer in the late 1960s found that the consumers in the target countries actually preferred younger animals. While this was initially resisted by the shippers, change occurred progressively. The study also found that the market preferred local fat-tailed sheep over the Merinos from Australia. This led to the department introducing the Awassi breed into WA. While there was resistance from the wool industry, which saw a potential problem of the introduction of black fibres into the wool, the breed was established and now contributes to the live sheep trade. There were problems of high death rates during shipping. The problem of the behaviour and management of sheep in export feedlots and during shipping is discussed separately below, with details of the import of the Awassi breed.

Other focused investigations
The management of sheep grazing saltbush pastures was studied. It was found that a 50:50 saltbush:dry feed diet gave far better production than saltbush or dry feed alone. Extensive work on the management and feeding of sheep in drought years was done, resulting in detailed advice being available to farmers during drought.

Experiments with sheep subjected to very cold conditions immediately after shearing showed that a plastic cover was sufficient to protect them from death, but if they were uncovered they could only maintain body temperature for about 10 hours.

It was demonstrated that while zinc was an essential element, toxicity could be developed as had occurred in the United States. Trials using stubbles for sheep feed showed that location, species and variety could all affect the digestibility and chemical composition of the straw. Researchers were also checking on the likely causes of lameness among young sheep fed cereal grain for long periods. It appeared that the problem was due to lack of calcium in the diet.

Research in 1986/87 showed that an additive, flavomycin, could increase wool growth by as much as 20 per cent without increasing fibre diameter. The additive could also increase liveweight gains by up to 30 per cent, according to the report. The department lodged a patent application for the use of this additive. Progress in commercial development of devices which slowly released the additive to grazing animals made use of this technology by wool producers technically feasible.

Flystrike
The primary blowfly (Lucillia cuprina) entered Australia in 1913 but was not detected in WA until 1934. Its wide distribution at that time indicated it had been in the State for some time. Its presence had a major impact on sheep management and breeding. In favourable conditions for the fly there were heavy losses in pastoral areas where flocks were scattered and intensive management was not possible.

In the agricultural areas early control relied largely on selecting plain-bodied sheep, crutching and strategic shearing to reduce or eliminate areas attractive to flies. Synthetic insecticides after World War II made control easier, aided by the promotion of the 'Mules' operation by the department after 1950.

This operation involved slicing off the loose skin and the associated wool from the breech, which was the major area of flystrike, leaving a bare area which was not attractive to flies. Later, the activities of animal rights groups overseas opposing this operation and stimulating a boycott of wool from mulesed sheep, caused the department to decide to operate internally without using mulesing and to focus on breeding sheep less susceptible to flystrike. The industry has been ambivalent on the use of mulesing in more recent times. In southern areas it is difficult to manage large flocks without using the operation.
Strategic extension
A major innovation in response to collapse of the wool market in 1990 was the development of a Wool Industry Strategic Extension Program. This was to extend the immediate and long-term implications of wool industry changes to growers. To support this program 33 Farmnotes were prepared and distributed to 190 advisers and private consultants from December 1990 to May 1991.

A computer model was developed to help with vital decisions such as how many sheep to run in a paddock, and how much phosphate fertiliser to apply. The model looked at prices and costs and calculated the point of optimum return based on wool production as influenced by the effect fertiliser rate and stocking rates have on pasture production.

Animal Breeding and Research Institute
At the new Animal Breeding and Research Institute the main studies were a comparison between Merino strains and, in a cooperative project with Merino breeders, breeding higher fertility Merinos. As a result 450 potentially high-producing sheep were transferred to the institute as foundation stock. The institute also carried out work on embryo collection and storage which advanced that process. A pilot reference scheme for stud Merino rams was also developed. This program systematically tested young rams from many studs against reference sires, which allowed researchers to accurately compare rams from different studs. The establishment of the sire referencing program was controversial but the results revealed important issues which the industry could not reject. Demand from other states resulted in valuable links with the WA scheme which identified rams of higher breeding value which might otherwise have escaped industry attention.

By 1984 the institute had five registered studs, Bred to Breed studs and Body Weight studs. Both types had a horned and not horned (polled) selection. The fifth stud was the base flock. These flocks provided semen back to the participating studs and the outcomes were followed.

Research was also undertaken to determine if there was a genetic basis for the production of tender wool in Western Australia. This indicated that fibre diameter distribution is a heritable characteristic. Work on ewe and weaner nutrition and immunisation to increase fertility was also undertaken.

Beef cattle
Early work with the beef industry in the south centred around feeding and health control to achieve a high reproduction level and the finishing of young stock for market. Feeding of heifers for growth to achieve early mating was also important. The available autumn pasture in the South West is determined by the length of the growing season. The cold conditions and shorter day length limits growth from mid-May through to June or early August and can result in limited high-quality pasture in years when the autumn break is late. Normally there is a flush of feed in spring. Ideally cows with calves at foot should have a good food supply available at the time of peak demand, when the calf is four to six months-old. This requires autumn calving. However, this could not be undertaken without considering the effect of autumn feed conditions on late pregnant breeders, the one-year-old replacement heifers, and yearling cattle being prepared for turnoff at 20 months of age.

These issues interacted with methods of fodder conservation and improved use of the spring flush. As a result, considerable work was carried out on finishing beef cattle in winter. This involved grain feeding where it was shown that urea could both provide a useful elemental supplementary source of nitrogen and could control the daily amount of grain supplement accepted by cattle from the self-feeder.
Increased fodder conservation in the spring and increased autumn feed were common issues for both beef and dairy cattle. Nitrogen fertilisers were extensively tested on both pastures and sown fodder crops. Work also covered areas such as the feed requirements of different cattle, diets, and degrees of fatness. In 2008, as part of the work of the CRC on Beef Production (centred on New England University) an experiment was being carried out to examine the energy cost of the mother in a beef production operation. Angus cattle were obtained which had been selected for high energy and low energy conversions or high and low feed efficiencies. It was shown that energy efficiency was an independently inherited character.

The challenge for the beef industry remained achieving reasonable on-farm prices, permitting the adoption of much of the technology from the dairy industry. Feed production systems could be the same but affordability was the problem.

In work with both dairy and beef cattle, baled and plastic-wrapped silage was shown to be superior to hay as a method of conserving high quality roughage. It was easy to store without deterioration if conserved properly. In common with the dairy industry there was a focus on reduction in the protein degradation of the higher protein content of lupins in the rumen.

Comparison of breeds and selection systems was undertaken in a long-term genetics and technology experiment at Wokalup Research Station. The results indicated that a well planned embryo-based selection system had the potential to double the rate of genetic progress in a breeding herd.

In the early 1980s the department tested the feasibility of developing a computerised auction system of selling as an alternative to auction in a market. This system was based on on-farm visual assessment of weight and quality. Having had independent assessment of the carcase weight and quality of an animal or a group of animals, the owner could then offer them for sale on the computer-based auction. If satisfied with the offered price he sold, if not he withdrew the animal(s).

In line with the policy of developing computerised models for complex estimates, a profit maximising beef cattle feeding model was developed in 1985.

**Carcase classification**

During the late 1970s and early 1980s there was a general thrust across Australia for the development of a carcase classification system for the beef industry. A system was established but research continued, seeking a system which gave a more accurate estimate of fat distribution through the carcase.

A carcase classification group was established to develop and promote the use of objective description for the marketing of livestock carcases and meat. The officers monitored classification in abattoirs, trained abattoir personnel in the system and sponsored the introduction of market development by specification and branding for both the domestic and export trades. A survey in March 1981 showed that the classification was firmly established at the retail and wholesale levels of the industry.

In 1986/87 a national industry body responsible for product description and quality assurance called Aus-Meat was established. It saw WA as having the most comprehensive and complete system for carcase classification of any state and requested the department hand over responsibility for monitoring various schemes. Two officers were seconded to Aus-Meat to assist with its early work.

**Kimberley cattle fattening and management**

As the industry contracted to the medium and higher rainfall areas following the recovery of wool prices in the early 1970s, greater attention was directed to finishing Kimberley cattle.

Early experiments with cattle brought south at six months, 18 months and 30 months of
age were disappointing. In general the cattle only achieved a fat score of 2 and after consuming a tonne of feed per head. However the quality of the beef improved over time and the results showed that Kimberley cattle would need closer management for longer periods, which would improve eating quality.

In 1985/86 further work was done on finishing Kimberley cattle. Fattening Brahman-cross cattle from the Kimberley was a means of increasing the productivity of Kimberley stations. Results showed that Kimberley cattle with a higher proportion of Brahman blood would fatten satisfactorily but at a slightly higher weight than southern steers. By 1990/91 the department was satisfied that a reliable system for finishing pastoral cattle in the south under both grazing and feedlot conditions was available. A full-scale extension program across all beef producing areas was conducted, resulting in a very large increase in the number of cattle brought south for finishing. In 1992 it was shown that finishing Kimberley steers was more profitable than finishing south-western steers.

A survey in the 1960s of the age of cattle slaughtered in Kimberley abattoirs found that 68 per cent were eight-tooth, 19 per cent six-tooth and 6.5 per cent four-tooths. Very few were killed at two-tooth or younger. This approach was doubtless due to a history of poor market access and the need to walk to the abattoir. It also showed a high percentage of dry cattle in the herd, which further reduced the percentage of breeders that could be run. By 1990/91 a long-term program on the Kimberley Research Station had shown that substantial gains in efficiency and profitability were possible through improved weaning practices. (See some additional detail in Chapter 7.) It was shown that weaning at both the start and end of the dry season increased branding percentages from around 45 to 85 per cent. In addition, cow mortalities were reduced from around 18 to 9 per cent. Breeding from Brahman bulls increased the growth and survival of calves. Application of this management practice had the potential to revolutionise the Kimberley industry.

Experimental work in the early 1980s showed that with infusion of Brahman blood and improved fencing for herd control, it was possible to manage the herd differently. If mating was controlled, calves weaned (at least in part of the herd), the weaners trucked south for fattening (or fattened on irrigated pastures where available), the percentage of cows could be increased. While cattle tick still needed to be controlled, the eradication of pleuro-pneumonia and TB had removed constraints on transport. Weaning itself was an important management practice as it allowed cows to put on condition, resulting in higher fertility. Even weaning and turning off two-tooth or four-tooth steers provided big opportunities. This work was developed into a management strategy which in later years was adopted by a number of Kimberley cattle stations.

**Dairy cow nutrition**

A dairy pasture utilisation and production project was completed, with 86 per cent of surveyed producers rating the project as successful in helping to improve production. In common with the beef industry a large amount of work was carried out on the relationship between stocking rate, nutrition and fodder conservation in the dairy areas. This included the use of nitrogen for both early autumn feed and increased hay or silage yields. It was shown that, in many cases, higher stocking levels could be carried comfortably when large amounts of fodder were conserved. Even at relatively high stocking rates part of the conserved fodder was carried over, depending on season. In general, the stocking rates achieved were considerably higher than traditional stocking rates in the area. This had a potential to result in a major increase in net farm income and viability. This work was expanded and tested in a large grazing trial at the Vasse Research
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Station in 2008. This experiment was testing the production from dairy cattle at five stocking rates and five rates of nitrogen fertilisation of the pasture. The rates of nitrogen were 0, 0.5, 1.0, 1.5 and 2.0 kg/ha/day applied as urea. The five stocking rates were 1.25, 1.5, 1.75, 2.0 and 2.25 cows/ha. Interestingly, the paddocks were no longer responsive to phosphorus and were topdressed with adequate potassium. The cows required selenium and may have required cobalt. In wet years there was need for additional sulphur in the latter part of the season. On-farm the nitrogen application and stocking was determined by the amount of ground cover. A leaf area index of 2.5 seemed about correct for maximum production. The production from the herd was very high. When the pasture dried off the animals were boxed and run on pasture irrigated with underground water. This could be the future for the dairy industry.

Poultry industry

The poultry industry was slow to establish in WA but during World War II had enjoyed a very good period of high demand. In the late 1940s a graduate poultry officer was appointed. He found that egg production per hen was low and undertook a production-research-demonstration program for both the laying and meat industry to bring it up to modern standards.

Breeds for both the laying industry and the meat industry needed to be sorted out, as were the feeding regimes. For the laying industry the most suitably-bred bird was selected and the environmental impact on its performance demonstrated. The major environmental issue was light, and in the early years techniques for light management developed. Initially, meat birds were also selected but as the industry became dominated by two major companies they adopted their own breeding stock.

Feeding studies

The emphasis in feed studies was on sources of protein and the balance between protein and energy. Of the items tested fishmeal appeared to increase growth rate more than other protein sources and this was confirmed in feeding trials. Rations with an energy to protein ratio of 45 to 1 were favoured. These experiments showed that riboflavin was not deficient in the normal diet.

A problem of the bruising of chickens during the early stages of processing was examined in 1984/85. This was caused within 12 hours of entering the processing chain and almost certainly during the catching and transporting process. Work was also undertaken on the use of a protected enzyme in increasing the efficiency of feed in broiler rations.

In trials on laying birds there was no comparable response to fishmeal. In contrast to the result from meat birds, experiments with layers showed riboflavin was deficient in the ration. With layers there was also an advantage of including green feed. Following this early work a full range of feeding trials was carried out.

With the later advent of lupins as a source of protein to replace the normal animal protein meals, particularly meat meal, experiments were carried out on the use of lupin meal. One special issue was the effect of lupin meal in increasing the moisture content of droppings. It was felt that if this could be overcome the use of lupins would be greatly increased.

Differences in the lupin meal from different varieties were tested as lupin meal became a major component of feed. The conclusion of this work in 1984/85 showed that lupin seed of either species was a suitable replacement for all or part of the meat meal in a layer ration. Another conclusion was that low nutritional density reduced egg production. Field peas were also tested as a protein source for layers. An algae which originated in the Pink Lakes near Esperance was also tested as a possible alternative yolk colour additive. A preliminary trial tested rapeseed meal from a new variety but it
appeared to slow growth rates on chicks from day-old to six weeks. Some serious disease problems also occurred, which are dealt with below.

Some disruption of the industry occurred when legislation to introduce quotas was implemented in 1971 to control over-production. Pressure for deregulation grew in NSW and controlled egg marketing was abandoned in that state in 1989, which meant deregulation in WA was inevitable.

**Pig industry**

Initially the work by the Intensive Industries Branch focused on diets which would achieve the low fat carcases sought by the consumers. Lupin varieties and species were tested as alternative protein sources to meat meal. As they became available, particular interest was directed to work testing lupin kernel meal and other factors influencing carcase quality. A sire referencing system was established in the pig industry.

Separately the Animal Health Laboratories confirmed that the industry in WA was carrying a severe form of Atrophic rhinitis. This caused twisting of the snout and bleeding and could affect growth.

In 1984/85 the group continued providing a service to industry by testing pigs for growth rate and depth of fat and providing a selection index. Almost 9000 pigs were tested under this program.

An investigation of a dietary enzyme response was initiated. In this investigation, growing pigs were fed restricted diets, including a protected dietary enzyme. There was significantly improved feed efficiency and growth rate. Whether the growth rate change was due to increased energy or increased amino acid supply was examined.

Research reported in 1986/87 referred to the study of initiation of early puberty in young female pigs. It had been found that exposure of gilts to a mature boar stimulated puberty. An investigation was started to determine the underlying reasons for the boar effect.

**Apiculture**

WA was free of major bee diseases, and maintenance of strict quarantine was a vital service to the industry. Beekeepers were interested in having a subjective assessment of the cost of production by the Department of Agriculture. A subsequent survey indicated an average reduction in financial liquidity from 92 to 28 per cent in the industry over the past two years.

Investigational work by the Apiculture Section focused on pollen quality, fertilisation opportunities and flora regeneration.

Queen bee production was also a major activity. The queen bee unit was the largest in the world, due to the disease-free status of Western Australian bees. By 1984/85 the bee breeding program, which had been in progress for five years, had developed superior stock and distributing this to the industry.
Bees have a marked impact on the yield of canola.

The difference in quality between pollen sources was examined. In the later 1990s opportunities for arrangements to provide fertilisation services to agricultural industries began to emerge. Parts of the fruit industry were interested and the developing canola industry provided further opportunity.

**Animal Health: Infection, nutrition, management**

The animal-based industries and the use of horses for traction and transport were vital to the development of Western Australia in the early days of settlement. While the areas available for cropping were limited it was possible to run sheep on the extensive areas of shrub and grassland in the semi-arid pastoral areas and the Kimberley. In the south, care had to be taken with any use of the limited native pastures or scrub land due to poison plants.

**Inspection and regulation**

The sheep scab mite, *Psoroptes ovis*, entered the Colony early and its history provided evidence that a livestock disease could be controlled using a regulatory approach. The WA Government initially enacted a ‘Bill for the prevention and cure of scab in sheep’ in 1866. This was followed by the *Scab Act* which prohibited the movement of stock from infected areas without treatment. Sheep scab acts had been introduced in other states and had proved effective. Sheep scab was eradicated from WA by 1895.

Control of disease was generally restricted to the knowledge base brought from England. Most of the work of professionals was focused on inspection at the ports. A government veterinarian was employed before the establishment of the Bureau of Agriculture. This officer and his staff were incorporated into the Department of Agriculture in 1902. The senior veterinarian was then titled the Chief Inspector of Stock. The normal work of the Stock Branch in the first 25 years was concerned with the inspection of imported animals, either from overseas or from interstate, particularly control of cattle imports to prevent bovine contagious pleuro-pneumonia being brought in from South Australia. There was also routine monitoring of the occurrence of endemic diseases and problems such as pleuro-pneumonia and cattle tick in the north, and tuberculosis (TB) of dairy cattle, lice and tick on sheep and problems of internal parasites, particularly of sheep, in the south. Contagious abortion (bovine brucellosis) became endemic and was reportedly being controlled by adoption of recommended practices. Action required later suggests this was an optimistic assessment.

In the general context of the development of the Colony, the 1903/04 report of the Chief Inspector of Stock is interesting. The bulk of the inspection work was at ports, with large numbers of stock imported. However, there had been an export of heifers from Wyndham to South Africa. In the south, the poultry industry was described as backward; impaction was the main source of death of dairy cattle, and although there had been only one reactor to the TB test, the Chief Veterinary Officer felt that the problem was widespread and he recommended compulsory testing of all dairy cows. He reported that influenza was a big killer of young pigs, and in that year lambing was ‘indifferent’ following a long dry summer.
In very early days cattle could be brought down from the north as there were no ticks until later, and pleuro-pneumonia was not recognised or seen as a problem. The hygiene at the abattoirs was obviously of concern and the chief inspector recommended that a central abattoir be constructed to improve meat inspection and the general hygiene associated with slaughter of livestock.

In 1904/05 the chief inspector reported that another 2000 heifers had been exported from Wyndham to South Africa. He also referred to tick-infested cattle being supplied to a Kalgoorlie abattoir. They were to be held ‘near Southern Cross at Hines Hill’. This suggests some ignorance of the geography of the State. Pleuro-pneumonia had been reported in some herds in the Metropolitan Area but had been eradicated. A swine fever outbreak was eradicated with losses to affected herds.

In 1909/10 it was reported that 7500 cattle were exported live from the Kimberley. In 1913 there had been a rapid growth in the live cattle trade to Java and Manila. It seems likely that the export was more frequent but was reported only spasmodically.

Rinderpest
An outbreak of rinderpest in 1923 was a serious problem but through firm action it was successfully dealt with. The disease was first reported from Beaconsfield. It was diagnosed on 20 November, and on 21 November the Chief Inspector of Stock ordered the destruction of all affected animals.

On the following day he ordered the destruction of all contact animals and all dairies in the area were quarantined. There was a lot of ‘common’ grazing ground around Beaconsfield, and control of cattle was not easy. There was also some hostility from dairy farmers who objected to their cows being shot. A further outbreak occurred in Bassendean on 26 November. As a result, the quarantine area was extended to a 30-mile radius from Fremantle. On 4 December a control board was formed, chaired by a pastoralist and including veterinarians from both the Commonwealth and State. The board took control and decided to slaughter all cattle, sheep, pigs and goats within a mile of an outbreak. A further extension from Bassendean was found on Rottnest Island on 27 December. The last case occurred on the mainland on 18 December. Rottnest Island was restocked on 11 February.

Western Australia was declared free on 26 March 1924 and all quarantine restrictions were lifted. This rapid eradication doubtless resulted from a willingness to take decisive action quickly. It was a credit to the limited veterinary staff of the State at that time. The disease has not recurred in WA. This was the first occasion where the eradication of a livestock disease was the subject of cost sharing with the Commonwealth, although the Commonwealth share was not generous. It was later determined that the disease had entered Australia in stock brought by ship via Derby and Singapore and off-loaded at Fremantle.

Swine plague and swine fever
An extensive outbreak of swine plague was diagnosed along the Great Southern railway and branch lines in late 1927. A large part of that region was quarantined. The disease had been largely eradicated by the following April. Isolated infected properties remained and had to be dealt with individually. It was not mentioned again in reports and it is assumed it was eradicated.

Outbreaks of swine fever were reported periodically over the early years but were eradicated by quarantine and slaughter of affected herds. The last occurrence in 1942/43, was eradicated on a slaughter-out basis and involved the slaughter of 12 000 pigs.

The losses to individuals were substantial and resulted in a Pig Industry Compensation Act being passed in late 1942. A levy was charged on all sales of pigs and paid into a fund, to be used to compensate producers
when their herds were affected by outbreaks of specified diseases.

Swill from an army camp was seen as the source of the infection which caused the outbreak in 1942/43. As a result, swill feeding was prohibited and remains prohibited today.

**Disease control and research**

**Bovine tuberculosis (TB)**

While bovine tuberculosis, caused by *Mycobacterium bovis*, was not a serious production-limiting disease for beef cattle, tuberculosis in the dairy herd was a major cause for concern because it is transmissible to humans, particularly children, through the consumption of contaminated, unpasteurised milk. Testing for TB was continuous from the early 1900s but reactors continued to be found at much the same rate, indicating that the disease level had changed little up to 1940/41.

In 1942/43 assistance was given to farmers to free their herds from both TB and contagious abortion (bovine brucellosis). The scheme, designed to accredit herds free of these diseases, was promoted but was not successful.

A trial on TB eradication at Harvey in that year showed that of 809 cows tested there were 5.5 per cent reactors. Later tests on many of the same herds showed the elimination of reactors had reduced the number of new reactors to 1.1 per cent. In 1946, the amended *Milk Act* replaced the *Dairy Cattle Compensation Act* and provided for the compulsory tuberculin testing of all cows owned by licensed dairymen supplying whole milk to the Metropolitan Area.

The Act also provided for all reactor cattle to be destroyed. Compensation was paid at a rate of up to $40 per head. During the first year, 12 590 cattle were tested and 2876 gave positive reactions and were destroyed. The 1948/49 results (see Table 3) are startling to a current reader.

The initial level of reactors in the Metropolitan herds had been 40.5 per cent. TB testing continued to 1965/66 but the reactor rate did not change much. Hence, while the prevalence of TB had declined to relatively low levels, eradication was not achieved.

In 1965/66, in the manufacturing milk sector, 22 165 cattle were tested with a prevalence of 0.4 per cent. In beef cattle, 42 904 were tested with a reactor rate of 0.14 per cent. Overall, testing showed a satisfactorily low level of disease in the South West. In 1967/68, 70 cattle of unknown origin were condemned at metropolitan abattoirs and 40 cases of generalised tuberculosis from 15 stations in the Kimberley were seen at abattoirs.

Table 3 *Tuberculin testing of dairy cows – 1948/49*

<table>
<thead>
<tr>
<th>Herd Type</th>
<th>No. of herds tested</th>
<th>No. of cattle tested</th>
<th>No. of reactors</th>
<th>Percentage of reactors</th>
</tr>
</thead>
<tbody>
<tr>
<td>Metro Area herds retested</td>
<td>45</td>
<td>3 362</td>
<td>160</td>
<td>4.76</td>
</tr>
<tr>
<td>South West dairy areas</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Herds tested for first time</td>
<td>161</td>
<td>10 773</td>
<td>1 918</td>
<td>17.80</td>
</tr>
<tr>
<td>Herds retested</td>
<td>148</td>
<td>9 343</td>
<td>345</td>
<td>3.70</td>
</tr>
<tr>
<td>Wanneroo – herds for first time</td>
<td>4</td>
<td>542</td>
<td>221</td>
<td>40.79</td>
</tr>
<tr>
<td>Bunbury – herds for first time</td>
<td>6</td>
<td>655</td>
<td>90</td>
<td>13.74</td>
</tr>
<tr>
<td>Geraldton – herds for first time</td>
<td>4</td>
<td>235</td>
<td>9</td>
<td>3.83</td>
</tr>
<tr>
<td>Other areas – herds for first time</td>
<td>10</td>
<td>883</td>
<td>176</td>
<td>22.00</td>
</tr>
<tr>
<td>Total for herds tested for the first time</td>
<td>230</td>
<td>16 450</td>
<td>2 474</td>
<td>15.03</td>
</tr>
</tbody>
</table>
Between 1958 and 1967 on-farm tuberculin testing had been conducted by private veterinarians with no supervision. This was found to be unsatisfactory, so district veterinary officers (DVOs) of the department took control of the testing program.

A national bovine brucellosis and tuberculosis eradication campaign (BTEC) was introduced in 1970, resulting in many years of extensive testing by government and private veterinarians. Eradication was based on detection (using the tuberculin skin test for TB and serological tests for bovine brucellosis) and slaughter, with compensation paid to the owners of infected herds. Critically, tail-tagging was also instituted, to allow traceback to the farm of origin when infected cattle were detected at abattoirs.

This massive, nationwide, complex and very expensive campaign occupied animal health services for many years. It was operationally difficult and became logistically complex, especially in the north. A national eradication campaign of this magnitude had never before been attempted, but with the financial support of the cattle industry and State and Commonwealth Governments, it was very successful. The south of WA was declared provisionally free of TB in 1976.

After that date, disease detection in the south was based primarily on abattoir surveillance. However, surveillance information was lacking on some herds and some tuberculin skin testing of ‘at-risk’ individual herds (mostly neighbours of infected herds) continued. Known infected herds were kept under quarantine and movement out of them was controlled. At 30 June 1976, 12 herds in the Kimberley and northern pastoral area were in this category.

Tuberculosis eradication continued in the Kimberley and in 1982 restrictive controls were placed on store cattle moving into the southern provisionally-free area. After four years of the program in the Kimberley the prevalence was estimated, from abattoir samples, to be 0.02 per cent. In 1985/86 the program continued in pastoral areas. Cattle were tested on 29 pastoral stations and the disease was found on three stations in the Pilbara, where eradication procedures were put in place. Some isolated TB infection was also detected in southern areas and necessary action taken.

In 1986/87 the program focused on the fencing of pastoral properties known to be infected, and a year later was reported to be on track to achieve an ‘impending freedom’ status for the Kimberley by 1992.

Throughout the TB eradication campaign, the department’s Animal Health Laboratories at South Perth had given specialised support by classifying the lesions detected at abattoirs and determining their cause. Because of doubt about the accuracy of the current tests the mycobacteriology laboratory, led by Dr Debbie Cousins, embarked on a research program. This improved the culture of *Mycobacterium bovis* and the precise identification of the organism using DNA techniques.
Chapter 6 – Animal health and production

From 1986, the laboratory played a pivotal role in investigations into the cause of tuberculosis in sea lions and NZ fur seals at a Perth marine park. One of the marine park trainers subsequently developed tuberculosis following infection with the same organism. Then in 1990/91, sea lions and NZ fur seals died from infection with the same organism on the south coast of WA, raising the possibility of a spill-over of infection from infected cattle. Dr Cousins was able to show, after some years of research using DNA fingerprinting and other techniques, that the *M. bovis*-like strains in the seals were genetically more closely related to the human TB organism, *M. tuberculosis*. Hence, a transfer of infection from cattle was unlikely. Eventually, the organism responsible for tuberculosis in seals and sea lions was classified as a new species of Mycobacterium, named *M. pinnipedii*. Similar techniques were used to investigate the likely sources of infection when ‘breakdowns’ occurred in cattle herds around Australia, including a large outbreak in Victoria in 1991.

The laboratory became the National Reference Laboratory for Bovine Tuberculosis in July 1992, giving it an Australia-wide role in ensuring the correct identification of TB in samples collected at abattoirs as part of the National Granuloma Submission Program. During the many years of service to BTEC, the laboratory had forged a worldwide reputation for excellence and was made an International (OIE) Reference Laboratory for Tuberculosis in 1993, which led to many collaborative research and training programs.

In later stages of the campaign efforts were concentrated on the Kimberley and Pilbara, where infection rates were relatively high and eradication had proved difficult due to transmission of infection between adjacent cattle stations. Eventually, the WA Government acquired ownership of some problem stations and, with extra spending on fencing and watering, was able to retest and destock as required. The last reservoir of infection was in the Pilbara, Warrawagine Station, bordering the Great Sandy Desert. Abattoir traceback showed this station had a high TB infection level. After several unsuccessful attempts, the station was finally destocked (20 000 cattle were removed) between 1988 and 1990, and no new cases were detected in the Pilbara after 1991.

Many departmental DVOs were involved in the eradication of TB from the Kimberley and the Pilbara, and the program proved problematic for five Chief Veterinary Officers. On 31 December 1997, Australia was declared a ‘Free Area’ for bovine tuberculosis. The Tuberculosis Freedom Assurance Program (TFAP) was then instituted, based on laboratory confirmation of suspicious lesions detected in cattle at abattoirs. The program led to additional detections, the last cases being seen in WA in 1998 and nationally in 2002. TFAP was finally completed in 2006.

*Bovine brucellosis (contagious abortion)*

Contagious abortion, caused by infection with the bacterium, *Brucella abortus*, is a serious cattle disease that can cause abortion rates of up to 100 per cent, and is responsible for a relapsing disease called undulant fever in exposed humans. It had become endemic in WA after colonisation. Early reports stated that it was being controlled by adoption of recommended practices. However, when the department's veterinary pathologists produced a test in 1938/39 it was hoped it would be possible to free a herd of this problem. This did not occur.

In 1945/46 it was reported that contagious abortion had been present in almost epidemic proportions and it was hoped to be able to use the newly developed Strain 19 vaccine to vaccinate heifers in the following year. In 1947/48, vaccinations proceeded with good results. There was a steady demand for vaccination in the following years and large numbers of heifers were vaccinated annually. By 1957 the disease was ‘largely controlled’ by the use of Strain 19 vaccine. In 1961/62, 37 600 heifers were vaccinated.
By this time outbreaks were restricted to areas where animals had not been vaccinated or vaccinated irregularly. Vaccination had greatly reduced the prevalence of disease, to the point that by 1965 there was talk of a national eradication campaign based on test and slaughter of infected cattle. By June 1970, vaccination was restricted to calves between three and six months-old but 50 700 heifers were vaccinated in the previous year. A new vaccine became available for adult cows and 331 cows had been treated during the year. In the Kimberley, 7653 serum samples were tested without a specific reaction being obtained.

Later, in 1970, the national BTEC program was initiated, with campaign funds being available to pay compensation for slaughtered cattle. There was a complication in WA because the use of Strain 19 vaccine over the years meant that positive reactions had to be further tested to separate those due to active infection from those due to vaccination. During most of the 1970s the brucellosis eradication campaign saw massive numbers of animals bled for testing in the laboratory.

Part of the Animal Health Laboratories in South Perth was remodelled in 1973 to accommodate the large-scale testing required. When the program reached its peak in the late 1970s, field officers were submitting about 16 000 samples a week, where the rose bengal, complement fixation and the serum agglutination tests were conducted. The scientist in charge of the brucellosis laboratory later conducted research to help distinguish vaccinated and infected animals. As a consequence, the laboratory also used the indirect haemolysis test from 1980. The enzyme-linked immunoglobulin test was added in 1983. The laboratory conducted six million tests for brucellosis during the campaign (from 1971 to 1986).

By 30 June 1975, testing, slaughter and strict movement controls had reduced the number of restricted herds in WA to 307. A system of tail-tagging was instituted to allow verification of the status of animals being sold. The number of restricted herds was reduced to 239 by 31 May 1976, and there were 1141 herds certified as brucellosis-free in the South West. Field sampling and laboratory testing had indicated that the Kimberley area was free of the disease. An important decision taken by senior veterinarians in 1975 was to discontinue vaccination, based on the perception that the vaccine policy had sufficiently dampened the infection rate to permit eradication to proceed. Vaccinations fell from more than 65 000 in 1971/72 to about 2000 in 1976/77. Until 1978, nearly all testing had been directed towards ‘at risk’ properties adjoining infected herds. Thereafter, the first round of testing of all herds in the south of the State began. In the first year 3000 herds were tested and only 14 were found to be infected. The first round of testing was completed in 1981 and the second round by June 1984. Provisional brucellosis freedom was declared in 1986 (by which time $700 million, in 1986 dollars, had been spent on BTEC nationally). This was followed by a monitoring phase, involving bulk milk testing and collection of specimens from abattoirs, which lasted until 30 June 1990. Australia was able to declare freedom from bovine brucellosis in 1989, at a direct (total BTEC) cost of $840 million. This was an enormous achievement for the large numbers of government and industry people involved in the campaign. When the extra costs to producers (mustering and yarding improvements) were added, the national cost was estimated in 1992 to exceed $1 billion. Eradication was claimed on the basis of farm inspection and testing with abattoir traceback as a safety net.

Contagious bovine pleuro-pneumonia (CBPP)

The infectious bacterial disease CBPP was inadvertently introduced through Melbourne in 1858 and reached the Kimberley in 1897. As veterinary services developed,
restrictions were placed on the movement of cattle south from the Kimberley. Cattle were required to be free of both cattle tick and CBPP and shipped out of Broome or Derby, inside the tick areas.

Cattle from stations where there were no ticks and therefore no resistance to tick fever risked picking up tick and contracting the disease as they were driven to Broome or Derby. They could not be guaranteed free of CBPP and would not be acceptable to southern markets. Fortunately, in 1958 CSIRO scientists working on CBPP developed the complement fixation test (CFT) which subsequently was used to underpin a national CBPP eradication campaign. The campaign was based on station-by-station testing, slaughter of carriers detected by the CFT, and vaccination of remaining stock.

The national campaign for eradication of CBPP from the industry, started in 1961, began in the Kimberley in 1964. Departmental officers in mobile laboratories tested cattle on-site using the CFT. In 1965/66 about 90 per cent of the cattle branded in the east Kimberley were vaccinated. No evidence of the disease was found in the west Kimberley. The west Kimberley was declared a protected area into which cattle could not be moved except under permit. No cases of the disease were found in the Kimberley in 1969/70 despite extensive field blood testing. The last confirmed case came from Carlton Station in 1967.

The disease was considered to have been eradicated, and Australia declared freedom from CBPP in 1973.

Research facilities
Research became a significant role of the Animal Branch in the late 1920s. The appointment of the veterinary pathologist Dr Harold William (Bill) Bennetts in 1924/25 opened the door to study of the causes of some significant stock diseases. Moves to establish a veterinary laboratory were prompted in part by the 1923 rinderpest outbreak, when the absence of a local laboratory to verify the diagnosis was a distinct disadvantage.

Also in 1925 the department agreed to fund a study of the lifecycle of the buffalo fly, jointly with the Commonwealth. The aim was to provide a basis for attacking the pest which was causing considerable problems across northern Australia. No control measures were developed; the problem remained and was subject of a further intensive research program later.

Dr Bill Bennetts made a major contribution to disease control in WA and won an international reputation for his groundbreaking research.

Following his appointment, Bennetts worked with makeshift facilities at several locations, including those at the Avondale State Farm. However, it was not until 1947 that the specifically-designed Animal Health and Nutrition Laboratory was constructed at Hollywood, allowing Bennetts to work with satisfactory facilities until his retirement in 1959. A new Animal Health Laboratory was commissioned in 1960 with the opening of the department’s new premises in Jarrah Road (now Baron-Hay Court), South Perth.
Enterotoxaemia of sheep (braxy-like disease, Beverley disease)

The so-called ‘braxy-like’ disease caused substantial losses for stock owners on the Great Southern railway line. Poor transport and communications and lack of facilities made this a difficult problem because the animals decayed quickly after death. To overcome this problem, a field laboratory was established at Beverley, but this was only partially successful and by 1927 Bennetts decided to live and work on the affected farms. This gained the respect and trust of the farming community and allowed study of the disease at close quarters.

Bennetts was seconded to CSIR in 1927/28 to enable him to work full-time on the disease.

During 1928/29 it was shown that the disease was caused by the toxins produced by a massive increase in the population of an intestinal bacterium, believed to be Bacillus welchii. Subsequently, Bennetts named the causative organism Bacillus ovitoxicus (now known as Clostridium perfringens Type D). In 1929/30 the department decided to set up a laboratory at the Avondale Research Station to investigate the factors predisposing sheep to the problem and to try to develop a vaccine. CSIR contributed £500 ($1000) for laboratory equipment. Beverley farmers contributed £200 ($400) towards the building and the department funded the rest.

A vaccine was produced in 1930 based on specific (Type D) WA strains isolated by Bennetts. The vaccine proved successful. The problem was now understood and a prophylactic was available. Later work established the need for annual vaccination in areas prone to the problem.

The 1931 report records that the research on the ‘braxy-like’ disease had been successfully completed. Bennetts was credited internationally with the discovery of a new concept for disease development – diseases caused by the absorption of bacterial toxins. These studies and the success of an Australian vaccine galvanised much overseas research into similar conditions caused by clostridial organisms. Today’s farmers vaccinate their sheep annually against enterotoxaemia or ‘pulpy kidney disease’, as the problem has become known over the years.

Botulism (toxic paralysis)

In the 1920s the southern areas of Western Australia were largely free from contagious diseases of livestock. However, there was concern about the high incidence of botulism or toxic paralysis among sheep which developed a depraved appetite and ate carrion from large numbers of rabbit carcases caused by the onset of dry conditions and shortage of feed in the wheatbelt. A laboratory was set up at Meckering to study the problem.

South African work with cattle had identified phosphorus deficiency as a primary cause of depraved appetite leading to consumption of carrion. In 1933 the department advised farmers to use phosphorus licks but further work showed that the sheep were not suffering from phosphorus deficiency and the phosphorus licks were of no value. Work testing various supplementary feeding regimes showed that high protein diets gave control, indicating that protein deficiency was the likely cause of the depraved appetite. However, economically viable protein supplementation regimes were only partially successful. Water sources could be contaminated but could be easily ‘purified’ by treatment with lime. By 1928, Bennetts demonstrated that toxic paralysis was caused by the ingestion of a toxin produced by Clostridium botulinum, a bacterium growing in the carcases of rabbits and other animals.

The final solution came through a vaccine prepared with the help of Dr LB Bull of CSIR, which was shown by Bennetts to protect sheep from the toxin (botulinum) under field conditions. The achievement of higher protein summer feeds by the introduction of a legume and the control of rabbits were management options which became...
progressively available and reduced the prevalence. However, the risk of botulism was ever-present during the summer, when carrion eating was often unavoidable. Vaccination is now a routine form of protection wherever ruminants are grazed, worldwide.

*Mastitis*

Mastitis has always been an endemic disease of dairy cattle. In the early 1960s, a major survey of its incidence in the dairy herds of Western Australia was undertaken. For the survey, mastitis was defined as an inflammation of the udder, producing evidence in the milk. The survey showed that two in every five cows were affected. While the incidence ranged widely between herds, no herd was completely free.

Staphylococcal organisms were found in 32 per cent of cows and streptococcal organisms in 4 per cent. In 25 per cent of the herds examined, staphylococci alone caused the infection. In 3 per cent of the herds streptococci alone caused the problem. In the remaining herds both organisms were present. Some herds contained staphlococci from human sources that were very resistant to antibiotics. The bovine strains did not show this resistance. Wastage in the State herd due to mastitis and low milk production was about 20 per cent. The conclusion was that, because of mastitis and antibiotic resistance, the milking herd was replaced every four to five years, which meant that most cows did not reach their genetic potential. For these reasons mastitis was very expensive for the industry. Typing of the staphylococci obtained from the survey showed several herds carried a type that was very invasive to humans and a considerable number belonged to a group commonly associated with outbreaks of human food poisoning.

The continuing nature of this problem was demonstrated by the Dairy Industry Authority introducing compulsory testing of milk, and a price penalty for high inflammatory cell counts in April 1987. This followed testing by the department’s Bunbury laboratory, which showed a significant proportion of milk with cell counts over 50,000 cells per millilitre. Field officers were advised of those farms with a cell count of more than 50,000/mL and advice was provided to the farmers concerned. It was found that 20 to 30 per cent of all high cell counts were associated with mastitis organisms. This program resulted in a significant reduction in the problem.

On-farm management practices were developed to control the problem. Back flushing of the milking cups coupled with the development of a rapid test to identify affected animals reduced the spread of the disease. The treatment of dry cows was also an important part of the program.

The management practice of back-flushing and treatment of cows as they dried off was developed and heavily promoted by departmental veterinarians. A major program involving a large number of herds kept under continual observation over the milking period proved the effectiveness of these techniques and provided the vehicle for convincing industry of their value. By 2008, the processors who had taken over the role of the Dairy Industry Authority imposed price penalties for high milk cell counts, and cows with mastitis were immediately withdrawn from the herd. This has resulted in mastitis now being successfully controlled in the dairy industry.

*Liver fluke*

In 1987 liver fluke was detected in one animal at an abattoir. This was of concern because it was known that the snail host necessary for completion of the lifecycle of the fluke was already in WA. Through traceback to the farm of origin, officers identified other infected animals on the original property and neighbours’ properties. Control measures were implemented and in 1989 eradication was claimed on the basis of farm inspection and testing, with abattoir traceback as a safety net.
Footrot in sheep

There are two forms of footrot in sheep:

- ‘Virulent footrot’ in which the destruction of the affected hoof is progressive, produces lameness, severe production loss and sometimes death
- ‘Benign footrot’ in which a similar lesion is mild and transient and affected sheep quickly recover.

Both forms are highly contagious but only the virulent form has economic and animal welfare significance. Both forms were widespread in the South West Land Division in the 1940s, causing considerable production loss. Moves by the Department of Agriculture to eradicate footrot began in 1947, when it was discovered that quarantining the farm and culling affected animals before the hot dry summer would usually result in whole-farm eradication, provided infected sheep were not introduced in the meantime.

By 1953 a full-scale campaign to eradicate footrot, based on these principles, was underway. Properties in quarantine were only permitted to sell sheep for immediate slaughter. During the year ended June 1955, 92 properties had been freed but 130 remained under quarantine. In 1958, only 61 properties remained in quarantine as opposed to 97 in 1957. By 1960/61, 20 properties were cleared and only 16 were left in quarantine. However, in 1965/66 the program took a step backwards with an outbreak in the South West during the early summer. The seemingly uncontrolled spread of footrot continued until 1974 when the decision was taken to exclude benign footrot from the eradication program.

Research was conducted at the newly-created Albany Regional Animal Health Laboratory to develop a test to distinguish benign from virulent footrot. This was successful with the discovery, by microbiologist Dr L Depiazzi, of the degrading proteinase test in 1978. This allowed more accurate prediction in the field, which supported the decision to quarantine farms for virulent footrot.

The eradication program was continued into the 1980s, during which time the number of farms in quarantine for virulent footrot fell to almost zero. However, an influx of sheep from the drought-stricken eastern states in the early 1980s was followed by the discovery of a large number of infected farms. In December 1985 all live export holding yards were declared quarantine areas, which gave farmers an outlet for healthy sheep from their quarantined properties. Success was considered possible with the footrot eradication program in the mid-1980s. However, in 1988/89 the program was set back by a major outbreak in the high rainfall areas from Boyup Brook to Augusta; the number of properties under quarantine doubled to 113, which represented about 1 per cent of sheep farms in WA.

The Albany Regional Animal Health Laboratory continued to conduct research into virulent footrot, demonstrating the strong interaction between the strain of the transmissible agent (Dichelobacter nodosus) and the environment. These studies helped to explain the regional distribution of virulent footrot in WA, and highlighted the importance of limiting spread between farms in the different rainfall zones of the South West.

In 1997 the Albany laboratory was recognised as the Australian National Reference Laboratory for ovine footrot. The degrading proteinase test and its derivatives proved to be the definitive test for virulent strains across Australia and put the WA eradication program on a sound basis. The number of properties under quarantine for virulent footrot increased from 48 to 58 during 1998/99, largely due to increased surveillance conducted at abattoirs.

In 2003, sheep industry representatives agreed to provide majority funding and to be involved in managing the eradication program which, until that time, had been supported entirely by State funds. However,
a subsequent decision was taken to abandon the objective of eradication and to focus on control. Under this plan, the farmer, having reported the presence of the virulent form on his property, had his property placed in quarantine and was given advice on how to manage the problem. It was left to the farmer to decide if he was going to aim for control or eradication from his property.

In 1999, a benefit-cost study using the Value Chain Model showed that attempts to eradicate virulent footrot in the previous 20 years had had substantial positive benefits. The program was estimated to have saved the industry about $116 million, of which $70 million was direct benefit to sheep farmers and the remainder a spill-over effect for abattoirs, butchers and others. The control of virulent footrot was subsequently passed to the hands of sheep farmers, with supporting advisory and laboratory services being supplied by the department. The continued low prevalence of footrot-infected farms was seen to have continued economic benefits for the industry in WA.

*Red water in calves*

This disease had been experienced for many years but despite extensive investigations no solution was found, although *Clostridium welchii* was the suspected cause. During the mid-1950s the cause was identified as the organism *Leptospira pomona*. It was believed that pigs were a reservoir of the disease and that calves needed to be kept from contact with pigs. While today a vaccine is available, this disease was a source of considerable losses in the early days of the industry.

*Mycotic dermatitis (lumpy wool)*

The mechanism of infection by *Dermatophilus congolensis*, the causative agent of mycotic dermatitis in sheep was elucidated by CSIRO researcher D Roberts following his departure as a veterinary microbiologist at the department’s Animal Health and Nutrition Laboratory. Subsequent studies at the South Perth laboratory clarified the role of dipping fluids and management practices in the transmission of this disease. These findings resulted in development of effective control strategies which are still in use, although a vaccine was developed later.

*Caseous lymphadenitis (CLA, cheesy gland)*

CLA became an important disease of sheep in the early 1970s due to international trade restrictions placed on infected carcasses. A research team at the Animal Health Laboratories, working in collaboration with CSIRO and the Commonwealth Serum Laboratory (CSL), developed a commercial vaccine (Glanvac) against the causative agent *Corynebacterium pseudotuberculosis*.

This vaccine is now widely used throughout the sheep industry and royalty income from sales provides revenue for the department to continue to support sheep disease research. Epidemiological studies by the Animal Health Laboratory CLA team demonstrated the importance of shearing cuts and sheep dipping fluids in transmitting the disease so that the use of the vaccine in combination with strategic management practices has greatly reduced prevalence in Australian flocks.

*Internal parasites*

With the introduction of improved pastures in the South West came higher stocking rates and the inevitable problem of internal parasitism in cattle and sheep. In the early 1960s, Department of Agriculture veterinary pathologist Dr MR Gardiner developed a system for determining the significance of worm burdens based on faecal egg counts with total worm counts for different nematode species of sheep and cattle. The ‘points’ system developed was still in use around Australia in 2008. In the 1960s parasitism was considered a specific disease requiring diagnosis and treatment.

In the 1970s, research by department veterinarian Geoff de Chaneet concentrated on cattle nematodes. Over about 10 years, studies on the ecology and epidemiology of internal cattle parasites were conducted at
The results provided the basis for the control program recommended today. It was not until much later (after 2005) that drench resistance in cattle was recognised as a global problem, and new studies were conducted in WA.

The emphasis of parasitology research moved from cattle to sheep in the late 1970s. The 'summer drenching' program, developed in Victoria, was tested at Mount Barker Research Station, with confirmation in 1980 that it dramatically reduced the number of drenches needed for effective worm control. Drench resistance in sheep worms was first demonstrated in 1979. Within two years, resistance was being found on an increasing number of farms. This led to a drench resistance survey (the first of its kind in Australia) that ran from 1982 to 1984 and showed that resistance was present on most farms in WA. This led to the 'CRACK' campaign (each letter indicates an action recommended to combat drench resistance), a comprehensive extension program that changed the way stock owners managed the complex interaction between parasite control and chemical efficacy. Surveys in following years consistently found WA farmers to be well ahead of their eastern states counterparts in understanding this issue.

The 'bell-wether' role of WA in the development of drench resistance was confirmed with the first published report of Ostertagia resistance to ivermectin in 1992. Researchers in the department discovered that ivermectin resistance was most advanced in situations where very few drench treatments were routinely given. The concept now known as 'refugia' was developed. This identified the need to preserve populations of less-resistant worms to dilute resistant ones, so resistance levels remained low as the basis of sustainable (low drench resistance) worm control programs.

Using ecological research methods, the group was able to confirm that in our Mediterranean climate few worms survived on pasture over summer, so resistant survivors of summer drenching were the main source of future worm populations. Consequently, worms in summer-drenched sheep developed resistance more rapidly than those in undrenched sheep. In the early 2000s, a large-scale series of observations on more than 60 farms showed that summer drenching was not necessary in adult sheep, which led to a change to 'summer-autumn drenching' recommendations. The concept of monitoring parasite burdens in sheep using repeated faecal worm egg counts as the basis of drench decisions was successfully introduced.

Major research was also conducted on non-chemical (genetic) worm control, through the establishment by John Karlsson of the Rylington Merino worm-resistant flock in the late 1980s. Over 20 years of breeding for lower worm egg counts, these sheep developed the greatest level of worm resistance reported internationally, and were used for many research projects on genomic and other indicators of worm resistance in individual sheep.

In 2001, the department research worker managing this program, Brown Besier, was appointed program manager for parasitology in the Australian Sheep Industry Cooperative Research Centre. The significant funding provided by the CRC resulted in better nematode diagnostic tests, less-selective programs for drench resistance, and a national website for worm control information (WormBoss).

**Lice control in sheep**

For many years there was a requirement that lice be controlled on every farm and plunge and spray dips were universal. In particular, all sheep were required to be dipped off-shears. However the discovery of the transfer of dermatitis through the dips immediately after shearing caused modification of the requirements. Control was still essential and sheep could not be sold through a public stockyard if infested.
In 1987 a proposal was developed, and supported by industry with a financial contribution, to eradicate sheep lice. This proved to be impractical and the program reverted to control. A major reason for abandoning the eradication objective was the development of resistance by the lice to the chemicals being used.

Squamous cell carcinoma
Long-term exposure to sunlight of the bare skin resulting from excessive removal of skin during mulesing sometimes resulted in a cancer called squamous cell carcinoma. This was managed through an extensive information campaign to mulesing contractors explaining the dangers of such radical wool removal. The cause was identified by a research program funded by the Australian Meat Research Committee in the early 1980s.

Urinary calculi
The problem of urinary calculi (urolithiasis) in wethers and rams fed grains for prolonged periods was recognised when sheep became common in the wheatbelt. It was extensively investigated by department veterinarians and chemists. Untreated urolithiasis caused blockage of the urinary tract leading to rupture of the bladder. The disease was known colloquially as ‘water belly’. Most uroliths in grain-fed animals were shown to be composed of magnesium and calcium salts. Uroliths with other compositions, including silica, occurred less frequently.

Uroliths developed primarily because prolonged grain feeding caused a change in urine pH towards alkalinity. Combined with high urine specific gravity, crystals were formed and produced a blockage. Treatment for urolithiasis involved the relative acidification of the urine using ammonium chloride at 1 to 2 per cent of the dietary dry matter. Salt was then added to increase water consumption, diluting the urine. Although the problem is still encountered, most stock managers are aware of the risks and the treatments available.

Infectious laryngotracheitis (ILT) of poultry
In 1947/48 a serious outbreak of the viral disease ILT occurred in poultry. Investigation showed that it had been present in WA for some time and eradication was not an option. ILT (a herpes virus) grown on dried egg was made available to poultry producers as a vaccine in 1951/52. A vaccination program was implemented for the areas where the disease had occurred. Each area was then quarantined, which meant no poultry except day-old chickens could leave the Metropolitan Area. The disease is significant for the poultry industry to this day. It is a nationally notifiable disease and vaccination is widely practised.

Pullorum disease
In 1953/54 pullorum disease in poultry was reported. It was decided to aim at eradication and compulsory testing of all flocks sending eggs to hatcheries was introduced. All hatcheries had to be registered and were monitored for disease presence. Regular testing for pullorum disease in fowls continued after the original diagnosis. In one year 75 600 birds were tested, of which 4470 gave positive reactions. In 1969/70, 327 400 birds from 90 flocks on 14 breeding farms were tested. Thirty flocks proved to be infected; 2370 reactors were detected representing an overall infection level of 0.7 per cent. Deterioration occurred caused through franchised hatcheries obtaining all replacements from the eastern states. The disease was largely eradicated from commercial poultry farms but it occasionally causes mortalities in backyard flocks.

Cobalt deficiency (Denmark wasting disease)
Reports of wasting and deaths of calves at Denmark from an unknown cause were a serious concern, as the government was committed to developing a dairy industry in the district.
Investigation into the cause of the problem began shortly after Dr Eric Underwood returned to WA in 1930 from postgraduate studies in the UK. In 1933 it was reported that the investigation of Denmark wasting disease ‘which had been started in earlier years’ was continuing. Underwood and veterinarian JF Filmer were in charge. There had been some indication that iron may be involved. In 1934 the iron product (limonite) was analysed and the components tested on affected animals. This showed that iron itself was not involved but the active principle lay in the zinc group of elements. This group was further divided into those with and without nickel and the group without nickel was not effective in curing the condition. By 1935 it was shown that the disease was caused by a deficiency of cobalt. Cobalt had been a contaminant in the nickel separated from the original zinc group. The disease was cured by adding minute amounts of cobalt to the diet. Initially a very dilute solution of cobalt and nickel was prepared by the local chemist in Denmark and sold to farmers.

The Waite Institute in South Australia was asked to map the soils which appeared to be associated with the problem. The soil survey of some 15 500 acres was later made by the Plant Nutrition Branch of the department, with some assistance from the Waite Institute. It was shown that the acute problem was restricted to one soil type. Later it was shown that cobalt was low and at times deficient in many other parts of the South West.

Copper deficiency in sheep (enzootic neonatal ataxia, Gingin rickets)

A problem of lambs born without the full use of their hind legs, known as enzootic ataxia, had existed for some time at Gingin. The view in 1933 was that it was caused by the ingestion of some toxic principle by the mother during pregnancy. Feeding trials showed that phosphate and mineral licks were of no value. By mid-1937 the cause of the problem was identified as a deficiency of copper in the diet of the ewe. This was based on experiments conducted by Bennetts and analyses by the government chemist. It appeared that on some soil types the pregnant ewe could not obtain enough copper for the normal growth and development of the embryo, and the newborn lamb was affected by ataxia. The ewe was anaemic for the same reason.

It was also noted that wool appearance and production was improved on treated sheep. While the virtual absence of the problem in the control flocks did not allow the effect of copper on ataxia of lambs to be fully investigated, the effect of copper treatment on the anaemia of the ewes and the improved wool growth and character were clear. An experiment to determine the frequency needed for copper dressings of pasture was established.

Surveys of copper status of farm animals showed that mild deficiency was widespread and that ‘stringy’ or ‘steely’ wool was a reliable indicator of copper deficiency in sheep. The identification of copper as an important trace element in WA stimulated investigations of its use by plants. This led to the opening up of the vast areas of light land. Bennetts was credited with the discovery of another new concept, that disease could be caused by deficiency of a dietary trace element. This discovery led to greatly improved animal and plant production in other places, notably the UK and South Africa.

Copper deficiency in cattle (falling disease)

In 1936/37 a problem known locally as ‘falling disease’ of cattle was identified in the Margaret River area. High producing cattle would fall down and die from no apparent cause. This often happened at milking time, sometimes in the bale, hence the name ‘falling disease’.

Investigation over a number of years showed that animals in the affected herds were anaemic. In the field the disease was usually associated with the presence of drooping-flowered clover. As there was no evidence
that the condition was infectious, the effect of various nutrients was tested. The animals treated with copper were healthier and not anaemic, and farmers were advised to give their cattle a lick containing copper. In 1945/46 an experiment on an affected farm indicated that copper deficiency caused progressive atrophy and fibrous tissue replacement of the myocardium, leading to heart failure under stress. The problem disappeared from the district once the use of copper fertiliser became widespread. It was found that drooping-flowered clover was very tolerant of low copper levels in the soil.

Copper and cobalt deficiency (coast disease)
Following South Australian work, the effect of copper and cobalt on animals affected by 'coast disease' in South West coastal areas was tested. As in South Australia, treatment with both copper and cobalt cured the problem. The affected areas were mapped as they were identified.

In 1958, in work with CSIRO to survey the copper and cobalt levels in pastures it appeared that sheep have a capacity to store copper and could suffer from toxicity. Following detailed studies by the department in 1961/62 a general 'ill-thrift' of sheep was attributed to marginal copper and cobalt levels. Coast disease was subsequently shown to be due to a dietary deficiency of cobalt, which is necessary for the production of vitamin B12 in the animal. Cobalt treatment of pastures and vitamin B12 supplementation of animals cures the condition.

Selenium and vitamin E deficiency
A survey also showed critically low concentrations of selenium in some pastures in high rainfall areas of the South West. Although pastures did not appear to suffer, a number of conditions were seen in grazing stock. Poor body weight and wool production and a degenerative myopathy ('white muscle disease') in lambs were documented. Mortality rates due to white muscle disease were high in some areas.

Field studies by department veterinarian Brian Gabbedy demonstrated a production response to treatment in susceptible areas. Selenium deficiency was thought to be associated with ill-thrift and often myopathy in sheep grazing cereal stubbles. Autumn ill-thrift in weaner sheep was later shown to be associated with seasonal protein malnutrition and depletion of vitamin E. While selenium protected sheep from white muscle disease, it was vitamin E that gave protection from autumn myopathy in both selenium-deficient and sufficient sheep.

Phosphorous deficiency of cattle
A problem of poor production and infertility in cows at Manjimup was investigated during the late 1940s. Studies of phosphorus levels showed cows on dry feed had low levels through summer. Experiments showed that up to the hay stage the phosphorus content of pasture was adequate for a four-gallon-a-day cow, even on areas topdressed with only one hundredweight of superphosphate per acre. After the hay stage even topdressing a pasture with 4 cwt of super per acre did not maintain the phosphorus at a level which would support a cow milking one gallon a day.

In 1947/48 an investigation of a serious infertility problem in cattle in the Margaret River area identified a serious phosphorus deficiency on the basis of low phosphorus blood levels. This also occurred late in the summer with little time to recover before the demands of milk production. It was found that milking cows needed direct phosphorus supplementation to raise their blood phosphorus to a 'normal' level.

A new research station was acquired at Bramley just north of Margaret River in 1948/49 to concentrate on this problem. In 1953/54 it was reported that the phosphorus-fed group produced more milk and butterfat than the controls. In 1955/56 the increased production of the control group, now receiving phosphorus as a result of reversing the feeding regime, confirmed the value of phosphorus in increasing milk production in...
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the area. This result was maintained in the 1957 research program, further confirming the importance of phosphorus level for milk and butterfat production.

Deaths during transport of sheep during live export

In 1985 the Senate Select Committee on Animal Welfare, following an enquiry into the live sheep trade, indicated there were compelling reasons to terminate the trade, but allowed a continuation based on recommendations for improved husbandry and additional research. Department of Agriculture veterinarians then embarked on a program of research, led by Barry Richards and Richard Norris, that ran from 1985 to 1990.

Extensive studies were made of the causes of deaths among sheep exported live to the Middle East, and the results published in scientific journals. A synopsis was given in the Journal of Agriculture in 1990. The work showed that about 3 per cent of the sheep that left their farms did not reach their overseas destinations through rejection before export or death during shipping. Approximately 12 per cent of these were rejected in the assembly feedlot and 15 per cent on the wharf during loading. Of the remainder which did not reach their destination, 54 per cent died during the sea voyage and 14 per cent died during unloading at the destination port. An examination of the causes of death found that 47 per cent of those that died during transport at sea or unloading did so because they did not eat from the time they left the farm (the ‘shy feeder’ or inanition syndrome). A further 27 per cent died from salmonellosis, often preceded by inanition (six of every seven that died of salmonellosis were shy feeders).

The work also showed that sheep had low salmonella levels when they arrived at the feedlot, but progressive environmental contamination of the feedlot yards and sheds predisposed them to increasing infection. This work clearly identified salmonellosis and shy eaters as the major issues. Further work showed that the major factors predisposing to death during shipping were season, age, fatness and farm of origin. Deaths were higher in the second half of the calendar year, the death rate of adult wethers was three times that of hoggets, and fat sheep were more likely to die than lean sheep. Inexplicably, a small proportion of farms contributed most of the deaths, and the reasons are still unclear. By 2000, the annual industry mortality rate had fallen below 1 per cent due to the adoption of recommendations arising from the research.

Vitamin A and weaner sheep

In the late 1940s experiments on digestibility of cereal hay showed the greatest yield of digestible nutrients occurred at the late milk to early dough stage in wheat – two to three weeks after flowering.

This work also showed that only ‘greenish’ coloured cereal hay had sufficient carotene to be a source of vitamin A. Dry pastures and cereal grain were virtually devoid of vitamin A or carotene. Based on the knowledge that animals at birth have no vitamin A in their livers, there was an interest in the vitamin A content of the colostrum of ewes following a dry summer. It was found that the level was only one quarter of the
‘normal’ level produced on green feed. Experiments testing the effect of this on lamb health were planned as it was known that calves with low levels of vitamin A were predisposed to a variety of infections. In the early 1950s a trial was carried out in which weaner sheep were treated with a large dose of vitamin A during the summer. There was no response to the treatment.

**Sheep fertility**

A long-term problem of sheep production in Western Australia has been of the low level of fertility of Merino ewes. Investigations into this problem on 20 commercial properties began in 1970 and suggested that the problem was related to the ewes not producing enough eggs.

It was found that for every 100 ewes served by rams an average of only 110 eggs were released. Higher protein diets with lupins were shown to increase the ovulation rate and fertility markedly. These experiments were conducted in 1974 and it was found that the ewes responded very quickly to lupin feeding. Feeding for only 14 days before joining with rams was as effective as 35 days. Once feeding ceased the ovulation rate and fertility quickly fell back to pre-feeding levels. The degree of increase depended on the nutrition of the ewes. The increase was higher where ewes were grazing on low-protein cereal stubbles as opposed to ewes grazing on subterranean clover-based pastures.

Two approaches were taken to genetically improve fertility in the Merino. One was the evaluation of the Booroola strain as a source of high fertility genes and the other was the mating of high fertility Merino ewes with rams that had been selected from ewes with a high fertility history. The progeny born within the high fertility flock were shown to be more fertile than those in an unselected flock.

In 1989 it was reported that two products, Fecundin and Regulin, had been shown to increase lambing percentage by up to 40 and 25 per cent respectively. Fecundin was a vaccine which affected the hormone balance of the ewe and increased the proportion of ewes releasing more than one egg – hence more twins. Regulin improved the conception rate and also produced more twins.

Keeping lambs alive after birth was also a major issue. On-farm studies in 1988 showed that management of the ewe during pregnancy, particularly maintaining her weight in mid-pregnancy, had a major influence on lamb deaths. It became possible to develop management programs to significantly reduce lamb losses and increase the efficiency of sheep production.

**Phosphate nutrition of Kimberley cattle**

A trial began in 1974 to study the effects of stocking rate and phosphorus supplementation on animal and pasture performance, using pasture established on pindan during the wet season and black soil plains pasture during the dry. Half the animals received a phosphorus supplement and half received salt only. Those animals given a monosodium phosphate lick consumed the equivalent of 4 grams of phosphorus a day during the trial. After the dry season of 1976/77, the low stocking rate group had no deaths when cattle received phosphorus, compared with 45-50 per cent deaths in the group on salt only. At the higher stocking rate, phosphorus reduced the death rate from 46 to 6 per cent. This work was part of the overall re-examination of the management of cattle in the Kimberley.

**Grain poisoning (acidosis)**

In 1993/94 work on the use of a feed additive virginiamycin was shown to reduce the gram positive bacteria in the rumen of sheep. These bacteria produced lactic acid, which could cause grain poisoning. This work was done in partnership with the Wool Research and Development Corporation and Smith-Kline Beecham. Its use in Australia was of value to the feedlot industry. Normal management practice required grain to be mixed with hay to avoid the problem and substantial mixing costs could be saved.
Plant related diseases and poisons

Native poison plants
One special part of the department’s early work was the identification of naturally-occurring poison plants which had a devastating effect on the livestock industries. There are more than 180 poisonous plants endemic to Western Australia. It is therefore not surprising that from early colonisation, poisonous plants have had a profound influence on agriculture. An extreme example is that between 1833 and 1840, more than half of the Colony’s livestock (sheep, goats, cattle and horses) were poisoned.

Many of the early poisonings were caused by shrubby legumes in the genus *Gastrolobium* (the genera *Gastrolobium* and *Oxylobium* were combined into the single genus *Gastrolobium* in 1987). Unfortunately the European colonists believed all legumes must be nutritious and they saw native animals eating these plants without effect, so made little effort to stop their stock eating them. It is now known that 34 of the 47 *Gastrolobium* species in Western Australia are toxic.

The department worked to identify which plants were toxic, define the problem, develop prevention and management procedures and inform the rural community of the details. As a result in the last 80 years poisonings from native plants have become only occasional. The first publication on the subject was the Department of Agriculture Bulletin 32 written by A Morrison in 1909. Bulletin 69 was written by DA Herbert in 1921, and revised by WM Carne, CA Gardner and HW Bennetts and published as a second edition in 1926.

After his appointment in 1924/25 the veterinary pathologist Bennetts worked on the toxicity of the *Gastrolobium* and *Oxylobium* species which were the most common of the native poison plants. He carried out tests of some 17 native plant species, 11 of which belonged to the *Oxylobium* or *Gastrolobium* genera. He commented that the toxic principle of *Oxylobium parviflorum* was water soluble and he expected that to apply to the *Gastrolobiums* as well.

He published a definitive paper on the subject, co-authored by CA Gardner, the Government Botanist. The toxic principle was later shown to be sodium fluoroacetate (later manufactured as 1080 for rabbit control). In the 1965/66 report studies of prickly and crinkle leaf poisons were reported. It was shown that increased metabolic activity in the plant following rain produced the fluoroacetate which made them toxic. They were likely to remain toxic for up to two weeks after such an event.

Bennetts and Gardner collaborated over 30 years in studies which defined which plants were poisonous, the clinical signs and any gross pathology produced. This work was reported in 1956 in an authoritative book, *The Toxic Plants of Western Australia*. *The Toxic Plants of Western Australia* dealt mainly with native and introduced plants where poisoning followed unknowing or accidental exposure of livestock. Knowledge of these types of plant poisonings was further increased by the studies of MR Gardiner, published between 1960 and 1976, and the Government Botanist, TEH Aplin, who published 35 papers between 1964 and 1984. Aplin expanded the scope of poisonous plants to include poisonous garden plants and toxic cyanobacteria and oestrogenic compounds which caused the problems in livestock grazing pastures dominant in these clovers. (See under Clover disease.)

In 1906 the State Botanist recorded an investigation of cattle losses in the Ashburton district. These proved to be due to a shrub of the Indigofera genus which he named *Indigofera boviperda*. Analysis of material in Perth showed the plant contained a toxic alkaloid. With so much work of this type needed it was surprising to read that the position of botanist (together with the
assistant entomologist and bee expert) had been abolished by mid-1906.

An unusual poisoning case was reported when 25 cattle died after being forced to eat radish as their major food for about five weeks. Work was also proceeding on characterising the poison in the *Isotropis* spp. Bracken fern was known to cause numerous deaths among calves in the South West but apart from avoidance, little work was done.

In 1989 it was reported that unexplained sheep deaths in the eastern wheatbelt were found to be due to oxalate poisoning. The plant responsible has been identified as the slender ice plant and chemists found that it contained 18 per cent soluble oxalate in the dry state.

**Subterranean clover infertility (clover disease)**

In 1942/43 an investigation began into the cause of dystokia among lambing ewes. This was the first reference of the major problem which became known as subterranean clover infertility, or clover disease. Apparently a number of cases had been reported in the previous two years but the extent of the problem had not been appreciated.

On first analysis it was thought it may be due to large lambs and ‘fat, sluggish ewes’ grazing on luxurious subterranean clover pastures that were understocked for the seasonal conditions. Also, eversion of the uterus was observed among ewes that had lambed, and wethers developed what was called a ‘high tail’.

Identifying the cause and treatment of the problem became a major project. In 1943/44 a team made up of EJ Underwood, HW Bennetts and FL Shier was formed to attack the problem. Bennetts explained the clinical signs by showing that pregnant ewes developed cystic lesions in the uterus, lactation was induced in wethers and maiden ewes, and some wethers developed severe hyperplasia of bulbo-urethral glands of the urogenital tract (causing the tail to be raised). No solution was found and the disease occurred in increasing numbers of animals in 1944/45.

While the cause had not been identified, it was accepted by this time that it was due to a hormone imbalance. In 1945/46 Underwood and Bennetts showed that similar lesions were produced in mice and guinea pigs fed an extract of subterranean clover. They then reproduced the disease in sheep by prolonged feeding of the synthetic oestrogen, diethylstilboestrol. On this basis it was concluded that subterranean clover contained oestrogen or an oestrogen precursor.

The program was now being directed by a team made up of CSIRO, Department of Agriculture and University of WA personnel. The program sought to identify the substance and examine differences in the toxicity of subterranean clover cultivars from different soil types and locations. The type of fertiliser and the toxicity of mixed pastures (with a balanced mixture of grass and forbs) and subterranean clover were also examined.

In 1947/48 it was reported that the infertility problem in sheep could be managed in the field by additional cropping and a focus on balanced pastures. Investigations continued to elucidate the chemical involved and its effects on the animal.

Investigations were continued in 1948/49 aimed at isolating and identifying the phyto-oestrogenic substances, the impact of environment on their production, distribution through the plant and their long-term effects on the breeding capacity of the ewe. The isoflavones thought to cause clover disease were finally identified as genistein, biochanin A and formononetin, when advances in analytical techniques improved the capacity to identify them.

The effective dilution of phyto-oestrogen intake resulting from balanced pastures and improved sheep management greatly improved the situation. While occasional problems were reported, clover-related infertility was no longer considered a
significant field problem by the end of the 1940s. The problem was not raised again until 1954/55 when incidents on areas where subterranean clover pasture had been recently established were reported. In 1958 attempts were made to isolate isoflavones from other subterranean clover cultivars without any real success.

In the early 1960s an examination of the effect of different subterranean clover cultivars was made by CSIRO using a ‘milking wether’ bioassay. This work showed that there were marked differences between cultivars and that the popular Yarloop cultivar contained high levels of the oestrogenically active substance. By this stage it was known that Dwalganup did not produce the classical disease if it was present as a component in mixed pastures. In 1965/66 increased problems on pastures dominated by the Yarloop, Dwalganup or Dinninup cultivars were reported. These reports continued from the recently developed areas over the remainder of the 1960s.

Advances in analytical techniques, including thin layer chromatography, were now available for more rapid identification of the nature and level of isoflavones. Using these techniques it was shown that formononetin was the potent isoflavone in all cultivars of subterranean clover. Unlike genistein and biochanin A, which were converted into non-toxic compounds in the rumen, formononetin was converted into a more oestrogenic compound, equol.

Some cultivars had low or very low levels of formononetin, and these were selected for a breeding program at the University of WA. Subterranean clover cultivars with a range of maturities and low formononetin levels were identified and commercialised. The first of these became available in the late 1960s and they were extensively sown over the following years. Traditional forms of clover disease were then greatly reduced and lambing percentages improved.

However, surveys in the 1970s showed that although clinical clover disease had virtually disappeared, the fertility of sheep grazing subclover pastures was still unacceptably low. The department estimated that poor conception rates were costing the State about one million lambs a year. Subsequent extensive studies conducted by CSIRO veterinarian Norm Adams in collaboration with departmental officers John Lightfoot and Keith Croker, demonstrated that long-term low intake of phyto-oestrogens caused anatomical changes to the cervix of ewes, making them infertile.

Later studies showed that successive generations of sheep exposed to low level phyto-oestrogen intake were eventually naturally selected for a form of genetic resistance. By 2008 it was clear that the clover disease story was not complete and that further work would be required to allow sheep to reach their full reproductive potential.

Lupins and lupinosis

Lupins were the cause of two major toxicities in Western Australia. The sandplain lupin (*Lupinus cosentinii*) was accidentally introduced into Western Australia late in the 19th century and soon became naturalised. In the 1920s the sandplain lupin and the introduced New Zealand blue lupin (*Lupinus angustifolius*) became widely grown to improve poor sandy soils and for stock fodder. Both of these lupins contained high concentrations of quinolizidine alkaloids, which under certain conditions could poison animals, causing a transient neurological disease named lupine poisoning. Farmers soon became aware of how this disease could be avoided.

The second major toxicity caused by lupins was the liver disease lupinosis, which became a serious livestock disease in Western Australia.

The first outbreak of lupinosis occurred in 1948, and outbreaks became annual events for the next 45 years normally following summer rain on dry stubble. Chief Veterinary Pathologist Dr MR Gardiner studied the disease extensively between 1960 and 1975.
He established that a fungus growing on dead lupin plants produced toxins that caused the disease. South African researchers identified the fungus in 1970, and named it *Phomopsis leptostromiformis*. In the 1990s a series of studies by WA Department of Agriculture plant pathologists established that the real cause of lupinosis was *Phomopsis* sp. with the teleomorph of this fungus being named *Diaporthe toxica*.

In the 1950s UWA plant breeder Dr AJ Millington initiated a breeding program, later taken over by Dr JS (John) Gladstones, to develop low alkaloid lupins as a crop plant. The first low alkaloid variety of *Lupinus angustifolius*, the narrow-leafed lupin, was released in 1967 and the second in 1970. These were followed by improved cultivars over the next decade (see Chapter 7 for details).

The area sown to lupins increased dramatically and was accompanied by an increase in the number and severity of outbreaks of lupinosis. The disease became a major limitation to expansion of the use of lupins. The worst recorded year was 1977/78.

Veterinary pathologist Dr JG Allen undertook the investigation of lupinosis and authored or co-authored 113 articles on the subject between 1975 and 2008.

During the 1970s and 1980s, extensive studies of the fungus by plant pathologist PMcR Wood, and biochemist DS Petterson in collaboration with CSIRO, identified the toxins produced by the fungus (linear peptides named phomopsins) and described many of their physiological effects. Allen also established that the causative fungus could infect the seed, raising concerns about the safety of the harvested grain and increasing the impetus to find a solution to this disease. At one time the role of copper, which was high in the livers of affected sheep, and the possibility that zinc may help overcome the problem were examined with various management approaches.

Gladstones turned his attention to breeding lupin varieties resistant to infection by the phomopsis fungus, starting in the late 1970s and bringing together a team that included plant breeders Dr J Hamblin and Dr WA Cowling, with Allen and Wood. In 1989 the first three phomopsis-resistant lupins were released. As the area sown to phomopsis-resistant lupins increased, the prevalence of lupinosis decreased. Today the disease is seen infrequently, and usually only in a mild form.

**Annual ryegrass toxicity (ARGT)**

The sowing of annual ryegrass throughout the South West during the early 20th century resulted in a higher quality and greater quantity of feed available to grazing livestock, and greatly increased the carrying capacity of pastures. Annual ryegrass became relied upon by the livestock industries, while ecological and habitat characteristics of the plant meant it became a highly successful weed in cereal crops. In 1968, a new neurological disease occurred in the Gnowangerup area in sheep grazing annual ryegrass pasture. An identical disease had occurred sporadically in South Australia since 1955. The disease was named annual ryegrass toxicity (ARGT).

Sheep suffering from lupinosis. Lupinosis was a major disease affecting the safe use of high protein lupin stubbles.

Lupinosis in sheep grazing low alkaloid commercial lupin stubbles had significant epidemiological differences to the disease in sheep grazing the bitter sandplain lupins.
From this small beginning, the number of outbreaks each year increased dramatically. Between 1968 and 1988, the number of new holdings reporting the disease doubled every three years, and between 1986 and 1991, 307 to 782 holdings reported outbreaks each year. By 1992 ARGT had been reported on more than 1400 holdings, and during the 1990s it was estimated that an average of 20 000 to 30 000 sheep and 230 cattle died each year. In some years there were considerably more deaths, with 80 000 to 90 000 sheep deaths reported in each of 1991 and 2000.

Department of Agriculture research into this disease has been extensive since the early 1970s.

Veterinary pathologist Dr PH Berry studied field epidemiology and pathology of the disease, plant pathologist Dr BA Stynes, together with other scientists in South Australia, studied the complex inter-relationships between the ryegrass plant, the toxigenic bacterium Rathayibacter toxicus (previously Clavibacter toxicus and Corynebacterium rathayi) and the nematode Anguina funesta that caused pastures to become toxic. Biochemist Dr P Vogel and CSIRO collaborators isolated and identified the toxins, named corynetoxins. Nematologist Dr IT Riley identified a fungus, Dilophospora alopecuri, that could potentially disrupt the life cycles of the bacterium and the nematode and developed it into a commercial biological control agent called twist fungus.

Microbiologist Dr Sue Sutherland developed an ELISA for the detection of the toxic bacterium that has been extensively used to ensure the export of non-toxic hay, and veterinary pathologist Dr Jeremy Allen conducted a national survey with South Australian colleagues that demonstrated widespread contamination of harvested grains with the toxic bacterial galls, thus identifying a significant risk that needed to be managed by the grains industry.

ARGT remains a significant disease of livestock today. Pasture and livestock management procedures have been developed to reduce the risk of disease, and the twist fungus has been spread over several hundred thousand hectares, but these measures have not been totally effective and the disease continues to occur and spread. The development of herbicide resistance in ryegrass has made control of the grass more difficult. A very effective, nematode-resistant ryegrass named Safeguard has been developed, but there are difficulties in getting it appropriately established and many farmers are reluctant to plant it because it has unknown potential to be another weed. CSIRO developed a vaccine that was very effective in preventing the disease in laboratory experiments but could not get funding to develop this into a commercial vaccine. Research to find an effective solution to this toxicity continues.

Pyrrolizidine alkaloid poisoning (Kimberley horse disease, walkabout disease)

Department of Agriculture reports through the 1940s noted that Kimberley horse disease remained a problem needing further investigation. In 1949/50 work was carried out in consultation with other states where the problem occurred.

The disease was initially examined on the basis that it was related to Birdsville horse disease, which occurred in Queensland. This was known to be caused by ingestion of a plant containing an hepato-toxin (indospicine). However, in 1952, observations indicated that a different plant toxin, pyrrolizidine alkaloid, was involved. In 1953/54, a joint investigation by the Northern Territory, WA and CSIRO confirmed that one cause of Kimberley horse disease was ingestion of the plant Crotalaria retusa. Subsequent investigation suggested that two other closely related species of Crotalaria could also contribute to the problem. In 1961/62 poisoning of cattle by Crotalaria crispata rather than C. retusa was identified in the Kimberley.
Other broadscale toxicities
Several other broadscale toxicities of lesser significance than those highlighted above have been encountered. These include perennial ryegrass staggers, phalaris staggers, phalaris sudden death syndrome, facial eczema, kikuyu poisoning, tagasaste staggers, tagasaste-associated leucodystrophy in neonatal calves, interstitial pneumonia and severe gastro-enteritis caused by reshooting canola, black soil blindness, sorghum toxicoses and photosensitisation in livestock grazing perennial and new legume pastures.

For all of these, department scientists have studied the diseases to identify the causes and developed prevention strategies that have been communicated to the farming community. In 2008 no less than three projects were underway to better understand and prevent livestock problems that might arise with three new pasture species. These are biserrula, an annual legume; tedera, a perennial pasture legume; and perennial panic grasses. All three cause, or have potential to cause, photosensitisation in grazing livestock.

Other new plant toxicoses continue to be diagnosed. The veterinary pathologists in the Animal Health Laboratories reported the occurrence of 25 new plant-associated diseases between 1991 and 2001. It is clear poisonous plants will continue to be important in Western Australian agriculture.

General activities

Awassi fat-tail sheep
Export of live sheep to the Middle East became a major industry through the 1960s and 1970s. Large older Merino wethers were exported almost exclusively until the early 1970s when a study showed a preference for younger animals and more particularly for the local Awassi fat-tail sheep, which were not available from Australia.

In the early 1980s the department decided to import this breed of sheep. Careful quarantine was essential to avoid the import of the disease scrapie into Australia. With part funding by the Australian Meat Research Committee (AMRC) an import protocol was developed to avoid this. This required the collection of embryos from sheep on a Mediterranean island and implanting these into ewes in Australia, to be followed by a long period of strict quarantine for the resultant progeny.

Awassi sheep were introduced after a meticulous quarantine program to provide Middle Eastern markets with the sheep they preferred.

When the AMRC withdrew its support an Australian-Kuwaiti company took over control of the quarantine and the associated development of the breed in Australia. The company also controlled subsequent marketing of the adults. Quarantine finished in 1991 and by 2005, around 100 000 sheep per annum were exported to selected markets in the Middle East. Fat-tailed sheep were highly desired in Middle-Eastern markets and buyers were prepared to pay a substantial premium. The Awassi is also a dairy breed but a proposal to foster a sheep dairy industry in collaboration with the University of WA was never realised.

A possible goat industry
Increased interest was reported in 1987 in the development of a goat industry as the basis of producing cashmere. While the national average production of cashmere from feral goats was around 60 grams a year and efforts to increase production by improved nutrition had been largely unsuccessful, there seemed a possibility of
some improvement with breeding. Captured goats from islands offshore from Carnarvon produced two to four times the amount of cashmere obtained from mainland goats. Breeding experiments were undertaken and production measured from the progeny of these island goats.

Goats also had a special advantage in that they ate saffron thistle. Around Geraldton about 200,000 ha of land was infested with this weed, which sheep do not graze readily. Even at low stocking rates, goats were reported to have grazed 96 per cent of thistles in a paddock. While all this was positive at the time, a collapse in the price for cashmere caused the work to be abandoned. A trial with goats in the pastoral areas failed for the same reason.