Paper 2.34

The history of bluetongue, Akabane and ephemeral fever viruses and their vectors in Australia 1975–1999

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The history of bluetongue, Akabane and ephemeral fever viruses and their vectors in Australia

with contributions from

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Developed as part of an integrated animal disease strategy by the Veterinary Committee
of the Standing Committee on Agriculture and Resource Management

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This document has been prepared to capture historical and unpublished scientific data on arboviruses in Australia. The contents of this document summarise the work and knowledge of Australian scientists and technicians over the last century. Many of the scientists are directly acknowledged in the text. However the tireless efforts of the laboratory staff of the Commonwealth Scientific and Industrial Research Organisation (CSIRO) laboratories at Long Pocket, Indooroopilly, Queensland and McMaster Laboratory in Sydney and the laboratories of each of the State departments of agriculture or primary industries deserve special recognition. Similarly, the efforts of scientists and technicians have been underpinned by the generous cooperation of numerous farmers and field personnel. This book was produced with assistance from the Australian Quarantine and Inspection Service (AQIS), Office of the Chief Veterinary Officer, Department of Agriculture, Fisheries and Forestry — Australia (AFFA); and Meat and Livestock Australia. Compilation of the book was coordinated under contract by AusVet Animal Health Services Pty Ltd. A large portion of the initial draft was prepared by Dr T.D. St George, under contract to AusVet. The contribution of many scientists to this work, including Chris Baldock, Glen Bellis, Alan Bishop, Angus Cameron, Bill Doherty, Trevor Ellis, Geoff Gard, Steve Johnson, Peter Kirkland, Lorna Melville, Mike Muller, Tony Postle and Dick Roe is gratefully acknowledged.

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Foreword

A number of insect or arthropod-borne viruses (arboviruses), such as bluetongue, Akabane and ephemeral fever viruses, are found in parts of the north and east of Australia. These viruses entered through the northern coastline, almost certainly by insect movement from countries north of Australia.

In the 1960s there was increasing concern about the apparent international spread of bluetongue virus. Because of the great vulnerability of Australian ruminants, it was recognised that arbovirus studies were needed to provide epidemiological facts. The north of Australia was chosen as the place to start because it appeared to be the source of some endemic viruses and at greatest risk from exotic introductions. National epidemics of ephemeral fever led to deliberate studies by the Commonwealth Scientific and Industrial Research Organisation (CSIRO). In 1969, details were published confirming the rapid passage of this disease from the Northern Territory into other areas of the country with climatic conditions suitable for the maintenance of the vector.

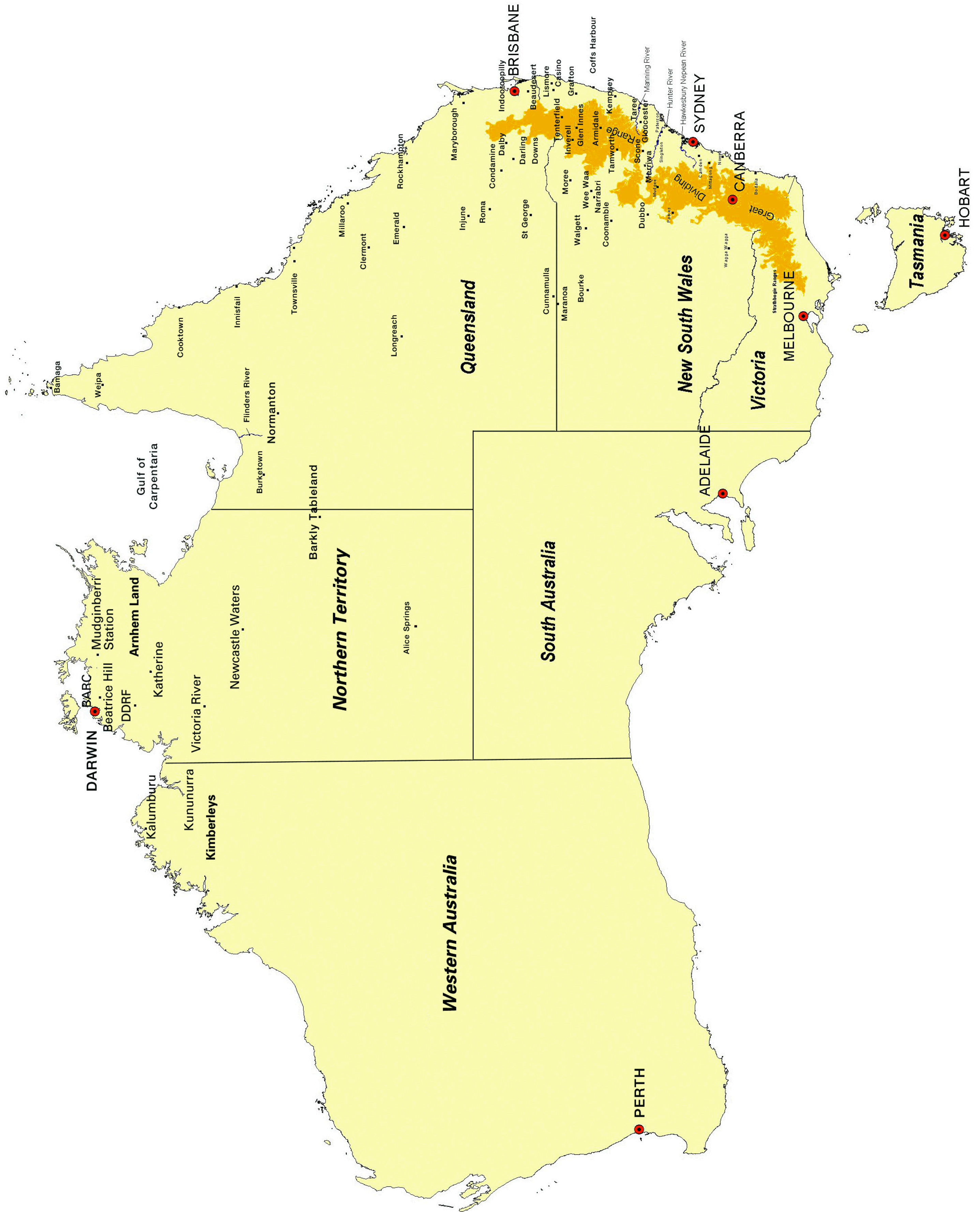
Beatrice Hill, about 100 km southeast of Darwin, was chosen as one of the first sites for sentinel herd studies. It had the advantages of a cooperating Northern Territory research station, road access to the edge of the subcoastal plains and abundant water buffalo, banteng, Brahman cross cattle, marsupials, waterbirds and insect activity.

The CSIRO scientists involved in setting up the studies had to endure tough climatic conditions and overcome the logistical problems of transporting equipment, insect collections and viruses from one end of Australia to the other. Their efforts bore considerable fruit, however, not least of which was the first isolation of a bluetongue virus in Australia, confirmed in October 1977.

These studies, together with the national sentinel herd scheme that developed concurrently, have proved invaluable in underpinning livestock exports by demonstrating that Australia has a credible scientific understanding of the distribution of these arboviruses.

This document traces the evolution of knowledge of the key livestock arboviruses in Australia, from superficial in the 1960s to the current situation where the natural histories of the vectors and viruses, and the reasons for variation in their annual distribution, are well understood.

R.W. GEE
Formerly Director of the Australian Bureau of Animal Health



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Chapter 1

Introduction

Arthropod-borne viruses (arboviruses) are spread by biting arthropod vectors, such as ticks, mosquitoes and midges. They multiply in a cycle — vertebrate > arthropod > vertebrate — usually in only a very limited range of species. Examples of arboviruses affecting humans are dengue and Murray Valley encephalitis. Arboviruses do not normally spread by contact in nature.

A number of arboviruses infect Australian livestock. Among these are bluetongue, Akabane and ephemeral fever viruses. These viruses have been found in parts of the north and east of Australia since at least 1936, having entered the country through the northern Australian coastline, probably by insect movement from countries to the north. Details about the occurrence, host range, transmission, pathogenesis and pathology and control of these viruses are given in Section 5.

The presence of these viruses, especially bluetongue, affects the export of live cattle, sheep and goats from Australia because a number of importing countries require serological testing, sourcing of exported animals from defined areas and/or restrictions on the ports from which the animals can embark.

Bluetongue virus was first detected in Australia in 1977 as a result of a deliberate research effort in northern Australia to identify arboviruses of significance to the livestock industries. This early work was often carried out in isolated locations, sometimes under difficult conditions and without the benefit of modern techniques. Nevertheless, most of these early research findings have stood the test of time and many are now considered seminal results.

When bluetongue virus was first detected in Australia, the initial response of Australian animal health authorities was to plan for an outbreak of clinical bluetongue in Australian sheep. Further studies were quickly started to define vectors of bluetongue virus in Australia and the distribution of antibodies to the virus. Specific policies and contingency plans were developed to deal with a bluetongue outbreak should it ever occur. These documents are available on the web site of Australia's National Animal Health Information System (NAHIS) under the section on AUSVETPLAN.[1] Most Australian experts in this field now believe that an outbreak of clinical bluetongue in the commercial sheep raising areas of Australia is unlikely.

This review outlines results of studies to identify arboviruses and their vectors present in Australia, and to determine their distribution and the factors that limit them. Most of the results of the virological and entomological examinations conducted over the last 20 years are now held in the National Arbovirus Database and can be accessed through the National Arbovirus Monitoring Program (NAMP).[2]

1 www.aahc.com.au/ausvetplan
2 www.aahc.com.au/programs/namp/index.htm

Many of the scientists involved in this research are recognised internationally for their contribution to arbovirus research in general, and bluetongue virus in particular. This review summarises their knowledge of the field aspects of Akabane, bovine ephemeral fever (BEF) and bluetongue viruses and their vectors in Australia. While the text does not contain citations, a comprehensive bibliography has been included.

Australian animal health authorities now have a sound knowledge of the distribution and seasonal nature of the various livestock arboviruses and their vectors. Of the arboviruses found in Australia, bluetongue is by far the most important because of its status as a List A disease,[3] which has implications for trade. A large zone in southern and central Australia has been continuously free of bluetongue virus transmission because it is unsuitable for the vectors. A buffer zone separates this free zone from an area in the north where bluetongue virus transmission sometimes occurs.

A joint industry–government funded project has started — the Northern Cattle Export Enhancement Project. The project aims to develop a climate-driven model based on the accumulated knowledge on bluetongue seroconversion obtained from sentinel herds over many years. The model will identify districts or regions from which cattle and sheep can be sourced for export with confidence that bluetongue virus transmission is not occurring in the district or region at the time.

The model will take into account topographical and climatic factors to provide a dynamic forecasting model to replace the static regionalisation system based on the maximum possible extent of bluetongue transmission. This should open the way for cattle exports from additional areas without risk of bluetongue virus transmission.

Arbovirus research in Australia has been supported by an effective collaboration between farmers, field veterinarians and laboratory scientists. That cooperation is now reflected in the joint financing of NAMP by the livestock industries, Commonwealth government and State and Territory governments.

3 A List A disease is one defined in the International Animal Health Code as having the potential for very serious and rapid spread, irrespective of national borders, which is of serious socioeconomic or public health consequence and which is of major importance in the international trade of animals and animal products.

Chapter 2
History of arbovirus research

Study of arboviruses has a long history in Australia. Initially, the interest was in human diseases. For example, dengue, which is spread by mosquitoes, has made many temporary entries into Australia. However, a major epidemic of another mosquito-borne disease — Murray Valley encephalitis — in the 1950s, and the isolation of the causative virus, provided a focus for research. This research brought Dr RL Doherty and his group's studies in northern Queensland to international prominence, because it led to the discovery of many arboviruses and their vectors.

Doherty's group was involved in studying livestock disease for many years, from the isolation of bovine ephemeral fever (BEF) virus in mice in 1968 until about 1977, when bluetongue virus was discovered. Dr Doherty enthusiastically cooperated in the first sentinel herd serology in 1971–72 — a cumbersome task because all the assays were carried out in suckling mice.

2.1 Early years

In the 1960s, northern Australia was a much more isolated region than today. A live cattle export trade from Darwin was not even considered. There were restrictions on the movement of cattle in the north because the area had not yet been declared free of pleuropneumonia. Dr RW Gee, the Chief Veterinary Officer of the Northern Territory, reviewed the perceived exotic disease threat to the area in 1970.

In the mid-1960s, serological surveys using newly discovered cattle viruses as antigens showed that the prevalences of antibodies to certain livestock viruses (bovine viral diarrhoea, infectious bovine rhinotracheitis and parainfluenza 3 viruses) were higher north of the Tropic of Capricorn. This was contrary to the prevailing view at that time that cattle in the north were substantially free of endemic virus infections because of low stocking densities.

In 1967–68, ephemeral fever spread from near Darwin to southern Victoria in a single summer, showing how quickly an introduced arbovirus could spread in unusual climatic conditions that were suitable for the vector's survival and spread.

Against this background, the Commonwealth Scientific and Industrial Research Organisation (CSIRO) established a new virology unit at Long Pocket Laboratories, Indooroopilly, Queensland, in October 1968, headed by Dr T St George. The unit's task was to delineate the endemic viruses of livestock and native animals in northern Australia. The aim was to develop expertise and knowledge of how the viruses already infecting livestock were spread, so that the findings and trained staff could be adapted to any emerging situation.

An entomology unit was already established at the CSIRO McMaster Laboratory in Sydney under the direction of Mr MD Murray and Mr AL Dyce. This unit had broadly delineated the biting midge (*Culicoides*) population of Australia, although its biology was still not well understood. Mr HA Standfast, a mosquito specialist, joined the

Long Pocket team in 1970 and Mr MJ Muller, a *Culicoides* specialist, joined in 1977. These appointments completed the coordination of arbovirus and vector research. Initially, there were no facilities for infecting livestock in insect-proof facilities, so most studies were directed towards understanding natural infection and disease.

2.2 The establishment of sentinel herds

Use of sentinel herds

Until about 1969, most information on viral activity was gathered through cross-sectional serological surveys. These surveys could only determine if the animals studied had been previously exposed to the viruses. A positive result did not indicate when or where infection had taken place. Animals could have become infected in one part of the country and then been transported elsewhere, testing seropositive several years after infection and hundreds or thousands of kilometres removed from the site of infection.

To understand the epidemiology of the viruses, and overcome the problems of cross-sectional surveys, a method for monitoring viral activity on a continual basis was required. Therefore, a network of sentinel cattle herds was established across Australia. To monitor virus infections, cattle in the sentinel herds were bled serially from six months to three years of age and the serum was stored. These stored samples could be used to test for exposure to any new viruses that might be found in the future.

Setting up sentinel herds

With the assistance of the chief veterinary officers of the Northern Territory, Queensland and Western Australia, field officers of each State and Territory assembled a small band of cooperators who, although aware that there would be no short-term benefit to them, nevertheless provided cattle and assistance. Each cooperator was asked to provide cattle for a limited number of years; one remained for more than 20 years. The sampling protocol is shown as Appendix 2. The objective was to obtain four samples a year, but in some areas cattle were handled only twice a year, limiting the frequency of sampling. The first samples were taken on 7 July 1969.

The animals selected as sentinels had to be born and remain in the location where they were bled. The fixed data (location, breed, sex, etc) were recorded in a form suitable for later computer manipulation. It was also intended that the variable data (age, date of bleed and serological results) would be computerised. However, the amount of data generated was soon beyond the capacity of the computers available and could not be analysed at the time.

In 1969, laboratory resources were very limited and there was no way to obtain and transport blood from remote areas. Some early samples therefore arrived in poor condition, due either to inexperience of the collectors or to lack of ice for cooling. At that time there was no information on the stability of neutralising antibodies to viruses in serum that was not kept cool. Aliquots of sterile serum of known antibody status were kept at temperatures ranging from $-100^{\circ}C$ to that of a hayshed through three months of an Australian summer ($> 40^{\circ}C$). Fortunately, neutralising antibodies are very stable. Therefore, their levels did not alter significantly, although the serum protein pattern disintegrated in a few days.

The scheme expanded in stages to include Papua New Guinea, southern Queensland, New South Wales and finally Tasmania, South Australia and southwest Western Australia by 1973. The southern herds became more and more valuable as time went on and arboviruses became the main focus of the study.

2.3 Early results of serology

Results from early research with sentinel herds indicated that parainfluenza 3 infection occurred at, or slightly before, the time maternal antibodies waned at 5–8 months. Bovine viral diarrhoea infection occurred between 6 and 18 months, whereas infectious bovine rhinotracheitis was more usual after 18 months. BEF virus appeared to be continuously active only in the far north of the Northern Territory, although the sampling regime was too erratic to confirm this. The chief veterinary officers and the cooperators were kept informed of the results and problems through a series of reports.

The first arbovirus results to be published related to ephemeral fever and Akabane virus. When serological testing (virus neutralisation) was carried out in 1971–72, Akabane virus was not recognised as a pathogen. Results indicated that most arbovirus infections occurred in summer or autumn and that antibodies to Akabane and other arboviruses often persisted for only six months after infection. Antibodies to Akabane were absent from cattle in Papua New Guinea and only found in one herd in southern New South Wales, at Griffith. Thus, an outline of the epidemiology of Akabane virus and its relationship to *Culicoides brevitarsis* in Australia was established before 1974, when its association with disease became known (see Section 5.2).

The accumulating serological results from tissue culture neutralisation tests on sentinel herd sera allowed a greater understanding of the movements of the virus. This enabled scientists to forecast in 1976 that an outbreak of foetal defects was possible on the New England Tablelands of New South Wales. Dr RE Everett and Dr OR Coverdale of the New South Wales Department of Agriculture prepared a reporting system before the outbreak began in the spring of 1976 and determined the aetiology in real time as a cooperative project with CSIRO.

Focus on arboviruses

In 1973, CSIRO senior management decided that the Long Pocket Laboratories should concentrate on ephemeral fever and other arboviruses. This resulted in a major reorganisation of the sentinel system. Instead of following individual sentinel animals for three years, a new group of susceptible animals was needed each summer.

Recruitment was at six months of age and an overlapping bleed between old and new groups was desirable. Group numbers had to be reduced because laboratory resources were stretched too far. From preliminary analysis of results, a group size of 10 animals was chosen. A larger group size was used on special occasions when a daily bleed interval was adopted for a short time to follow particular infections more closely. Where possible, the sampling interval in Australia was reduced progressively to one month. As the herds in Papua New Guinea were negative for ephemeral fever and Akabane virus antibodies, however, the interval between bleeds was not reduced. As far as possible, ephemeral fever serology took priority over other tasks. In 1974, a move to newly available technology reduced the requirement for serum from 1 mL to 0.1 mL per test, allowing a major saving in resources. These changes eased the field and laboratory aspects of collecting and processing blood samples.

2.4 Continuous arbovirus and entomology studies in the Northern Territory, 1974–76

An early analysis of the ephemeral fever serology indicated that seroconversion to ephemeral fever occurred most often in the sentinel herd at Beatrice Hill, southeast of Darwin. The research farm located at Beatrice Hill was on the western end of a subcoastal plain, between the Arnhem Land escarpment and the sea. The insect population was monitored at Mudginberri Station, a domesticated buffalo station around the midpoint of the plain. The team spent three weeks in each of the wet and dry seasons studying the insect population and perfecting trapping techniques. The daily pattern of insect activity was established through a 24-hour continuous trapping regime.

Ephemeral fever was not isolated from trapped insects. The site was not very productive in terms of arboviruses — two viruses were isolated with relevance only to indigenous species. The results showed that at least a year of sequential study was needed to understand the dynamics of the mosquito and *Culicoides* species that are vectors of ephemeral fever in the tropics. Other short-term expeditions, mounted quickly to find ephemeral fever vectors in the course of an epidemic, had failed.

The study showed the existence of separate species of *Culicoides* that were initially thought to be variants of *C. brevitarsis*. These additional species were *C. fulvus*, *C. actoni* and *C. wadai*. Partial understanding of their life cycles followed. One technique was to carefully examine the exterior of the *Culicoides* to see what parasites and microscopic traces of their breeding environment they carried.

The area around Beatrice Hill was much more suitable for a continuous study and was accessible by road all year. Dr RW Gee provided access to the cattle and buffalo herds. The demanding routine required the rotation of teams of entomologists and technicians from CSIRO. Collections were made for 81 weeks from October 1974 to May 1976, spanning two wet seasons. Earlier expeditions to Mudginberri had shown that much of the vector activity peaked before dawn and after sundown, so the collection times were tailored to match. The samples were sorted in the time between collections. Serum samples were collected weekly from sentinel cattle and buffalo. Target insect species were processed for virus isolation. Insects containing bloodmeals were sent to CSIRO McMaster Laboratory, Sydney, where Mr MD Murray determined which species the bloodmeal originated from. The main results took years to place in context.

The collection program was interrupted for a short time by cyclone Tracey in December 1974. All flying insects were blown away and breeding sites were washed out. The team in residence resumed the program when they had repaired the equipment.

The program's main achievement was to isolate ephemeral fever virus from a mosquito, *Anopheles bancroftii*. A further 93 viruses were isolated at the Long Pocket Laboratories from six species of mosquito and eight species of *Culicoides*.

During this study, a new tissue culture isolation method was introduced in parallel with the suckling mice traditionally used in Australia; it eventually replaced the use of suckling mice entirely. Until 1976, most viruses had to be sent to Dr RL Doherty at the Queensland Institute of Medical Research or to Dr RE Shope at Yale University, United States, to be identified. Using the new tissue culture isolation technique, bluetongue virus was isolated from a pool of mixed species of *Culicoides* collected on 27, 28 and 31 March 1975 and identified by the group at Yale in October 1977.

2.5 Discovery of bluetongue virus

Once bluetongue virus had been identified at Yale University, a bluetongue serum neutralisation test was rapidly developed and used to scan serum samples taken during 1975 (the period around the collection of the source insects). The most recent serum samples across the whole sentinel network were also scanned. Within eight days, the initial picture became clear.

The new virus (bluetongue serotype 20; BLU 20) had been present in Australia since 1974 and the current virus activity was confined to the Top End of the Northern Territory. This was of immediate importance to the regulatory officials. The rapidly mounted cross-sectional survey indicated that a very low prevalence of antibody titres to the serotype extended to northern Queensland and Western Australia. Thus, results from the sentinel herds indicated a much more restricted distribution than cross-sectional surveys. This discrepancy was later shown to be due to the use of different tests — serotype-specific tests were used for the sentinel herds and group-specific tests for the cross-sectional surveys.

All sera submitted from sentinel herds in November and December 1977 were tested by the specific neutralisation test and by a broadly reactive test for bluetongue group antibodies. The serum bank provided pre and postexposure serum samples from cattle with reactions to a bluetongue group test, which were sent to England for testing. Two serotypes, BLU 1 and one other, were found in cattle well outside the limited area where the sentinel herd system had identified BLU 20 infection, in spite of there being no clinical cases of bluetongue reported in sheep.

Cross-reactions with bluetongue serotypes 1 and 21 (BLU 1 and BLU 21) were later shown to be the cause of the reactions in serogroup tests. After BLU 20 was identified, it was realised that BLU 1 and BLU 21 had been circulating silently and infecting cattle subclinically for many years. While BLU 20 was restricted to the Top End of the Northern Territory, BLU 1 and BLU 21 were also found in Queensland, Western Australia and New South Wales. The BLU 20 serotype-specific neutralisation test only detected reactions in cattle sera from the Top End because this was where BLU 20 was localised. The immunodiffusion test detected all bluetongue serotypes (serogroup test) and therefore detected reactors in cattle in all the States where BLU 1, BLU 20 or BLU 21 occurred.

More detailed information was needed. The sentinel herd network was immediately expanded to cover the Barkly Tableland of the Northern Territory, because infection of this area could spread BLU 20 virus to the eastern states. The serology performed from 1977 to the present has not demonstrated any expansion of the infected area and shows that the serotype probably disappeared between 1980 and 1992.

There had been speculation as early as the 1950s that bluetongue virus would be present in a silent form in cattle in the north of Australia. It was first discovered in Australia during a search for the vectors of ephemeral fever virus. The discovery of bluetongue virus in Australia in 1977 was very important. Had the virus not been identified, importing countries would eventually have found antibodies to bluetongue viruses in cattle exported from Australia. A report from a foreign country that an undeclared agent of a List A disease was present in Australia could have had serious negative consequences.

2.6 Active search for viruses using sentinel herd bloods

Field collectors of sentinel herd blood usually submitted samples in the form of a clot that had begun to contract, showing clear serum around the clot. Some loose red and white cells were always found around the base of the clot. The practice was to remove the serum and discard the clot and loose cells. In 1976, a tissue culture technique introduced for detecting arboviruses in insects was applied to this cell debris from blood samples. A succession of viruses was detected and information obtained on lengths of viraemia. From 1978 to 1982, the technique was applied on a larger scale to find the additional bluetongue viruses shown by serology to be in Australia. The search had to be broad across the sentinel herds because there was uncertainty as to where and when these viruses were active.

The first isolates of bluetongue serotypes 21 and 1 were found in bloods collected on 23 February 1979 and 3 April 1979 respectively, from cattle in the Northern Territory. Once the serotypes were identified, the history of their distribution was determined from the serum bank. There was very little overlap (in time and space) between areas with these viruses and the area occupied by the national sheep flock. Serotypes 1 and 21 had been present in Australia since at least the beginning of the sentinel herd scheme.

By the time this active search for viruses was under way, Dr DH Cybinski at Long Pocket Laboratories, Brisbane, had developed methods to identify new arboviruses by group in a few months. As some viruses were new to science, they had to be sent to the relevant World Reference Centre in the United States or South Africa.

The direct culture of bloods also allowed the determination of the length of viraemia of Akabane and other viruses in daily bled animals. The definition of the length of viraemia in naturally infected animals is useful in developing protocols for trade in live animals.

A bull was infected with Akabane and the related Aino virus, indicating that reassortment of related viruses was possible. The blocking effect of bovine ephemeral fever virus by an unrelated virus was also demonstrated in a daily bled sentinel series.

The overall yield of viruses from blood clots was only 2% in the early years. However, the information obtained on the time and location of arboviral activity enabled later researchers to target particular locations and restricted seasons. By using cattle as the source, new viruses could be sought with far fewer resources, compared with those required for insects. Once a new arbovirus was identified by this means, its distribution could be determined very rapidly. Furthermore, while virus isolation from cattle is demanding, it requires much less scientific and technical expertise than identifying insect species before virus isolation can commence.

2.7 Final involvement of the CSIRO in the management of sentinel herds

In 1977, CSIRO management decided to cease their involvement in the management of sentinel herds and their responsibility for the serum bank. However, this move was deferred by the discovery of bluetongue virus: CSIRO maintained direct involvement with some sentinel herds until 1990. A National Serum Bank stocked with sera collected at random by the Department of Primary Industries, Canberra, replaced the CSIRO serum bank in 1979. In the event, no serological tests were done on samples held in the National Serum Bank because it was located far from the regional laboratories where scientists with a personal interest in using the sera worked.

Interest by individual States in the established sentinel herd technique continued. Herds near Darwin were maintained by Dr GP Gard, who established a new virus laboratory in Darwin, and by Dr PD Kirkland in New South Wales. In Darwin, Dr Gard and later Dr LF Melville systematised and improved the search for bluetongue viruses. The results are discussed in the description of bluetongue (Section 5.1). Dr Kirkland defined the epidemiology of Akabane virus in New South Wales.

Interest in the sentinel herd system was revived in the late 1980s, leading to the development of the coordinated national scheme (the National Arbovirus Monitoring Program, NAMP) in 1992. More recently, a national database has been established to assemble Australia's information on all the arbovirus monitoring studies conducted since the 1960s (Table 2.1).

Table 2.1 Number of sentinel herds used for monitoring arboviruses by State/Territory and year

Year	Qld	NSW	NT	WA	SA	Tas	Vic	PNG	Total
1969	4		2						6
1970	9		4	2				3	18
1971	11		4	2				3	20
1972	11	8	4	2				3	28
1973	12	8	4	2		1		4	31
1974	19	8	4	3	2	1		3	40
1975	18	12	5	3	2	1		3	44
1976	20	21	6	4	2	1		3	57
1977	17	26	6	4	1	1		3	58
1978	11	18	22	3		1		3	58
1979	4	4	8						16
1980	2	8	7						17
1981	2	7	7						16
1982	2	4	4						10
1983	2	4	3						9
1984	2	4	2		3				11
1985	2		2		3				7
1986	1	3	2						6
1987		3	2						5
1988		14	1						15
1989		27	5		4		4		40
1990	18	26	5	3	4	4	4		64
1991	17	24	7	4	4	4	4		64
1992	13	24	8	4	4	4	3		60
1993	15	21	7	4	6		4		57
1994	29	24	7	6	4		2		72
1995	29	25	7	6	4	3	4		78
1996	22	20	7	6	5	3	3		66
1997	21	22	9	6	6	3	4		71
1998	21	35	9	10	4	3	4		86
1999	23	29	9	8	4	3	3		79

Note: Blank cells indicate no samples were taken.
Source: National Arbovirus Monitoring Program database

CSIRO retained two herds for intensive studies of ephemeral fever and associated vector studies. In particular periods, cattle in one of these herds were bled daily for virological and biochemical studies, an incredible contribution by the owners, Terry and Mavis Hunt. This association continued up until 1990.

The few remaining herds yielded a large amount of information on the epidemiology of bluetongue, Akabane and ephemeral fever, which is covered in the sections on those viruses (Section 5) and has made Australia pre-eminent in knowledge of the natural history of these viruses and their infections. Almost every arbovirus that infects livestock in Australia has been found in the area around Beatrice Hill, indicating that this is probably a major focus of arboviruses entering Australia by mechanisms as yet unproven. Intensive monitoring at this location has continued under NAMP.

2.8 The insect light trap network

For about 100 years, entomologists had been studying insects that bite humans and animals in Australia, so the distribution of many flying insects that bite livestock was known. Many entomologists had made collections in locations scattered across Australia, concentrating their studies on flying insects that were vectors of viruses infecting humans. Some early identification of species was in doubt because certain species of *Culicoides* and mosquitoes had been given names that might not have been valid. Where museum specimens existed, the errors could be detected, but obtaining new material was preferable.

A three-year study of sentinel herds along the Flinders River in northern Queensland (1974–77) allowed the seroconversion of cattle to ephemeral fever and other viruses to be correlated with the presence or absence of suspected vector species. In the second year of this study, the sentinel sites were toured. The cooperating station owners were shown how to set and clear insect traps at suitable locations, and what supporting data to record. Early light traps were much less sophisticated than those available later. This pilot project demonstrated that locally based cooperators could trap insects on a routine basis. Unexpectedly, it was found that *C. brevitarsis* was active at an earlier time of year in the drier inland areas than on the coast. This indicated that the drier inland areas were suitable for the breeding of *C. brevitarsis* at restricted times of the year. Subsequent laboratory studies found that constant temperatures above 35°C restrict the development of *C. brevitarsis* larvae. Such temperatures occur in much of the northern Australian tropics between October and February and probably limit populations in these areas.

As resources allowed, the network was expanded by placing traps at existing sentinel herd sites. There was a reduction in activity in the 1980s, as with the sentinel herd monitoring, but an integrated national network has been re-established under NAMP (Table 2.2).

This technique of vector sampling was useful only for species attracted to light. When light traps are set over cattle in yards or in camps, they are more efficient in trapping species of *Culicoides* attracted to cattle. Other trapping methods were occasionally applied, complemented by intensive studies in selected locations. These included the use of truck traps, sweep netting, rearing adults from larvae in dung and, more recently, vacuum samplers. The expansion of the range of *C. wadai* down the east coast of Australia required more intensive monitoring by nets mounted on vehicles. Further details are described in the section on vectors (Section 4).

Table 2.2 Number of trapping sites for monitoring arbovirus vectors by State/Territory and year

Year	NSW	NT	Qld	SA	Tas	Vic	WA	Total
1975			2					2
1976		1	5					6
1977	8	2	11					21
1978	9	2	12				3	26
1979	6	1	10				6	23
1980	3	2	6				7	18
1981		17	12				5	34
1982	1	19	13				2	35
1983		7	4					11
1984	5		9					14
1985	12	4	17					33
1986	6		21					27
1987			3					3
1988			3					3
1990	40	9	51				12	112
1991	53	14	61				34	162
1992	49	7	55			36	29	176
1993	17	4	11	1		19	8	60
1994	27	9	20				10	66
1995	33	9	20	4	3	1	9	79
1996	28	10	18	4	3	3	7	73
1997	39	9	12	4	3	4	9	80
1998	40	11	17	4	3	4	11	90
1999	32	13	25	5	4	5	13	97

Note: Not all sites were in continuous use for the entire year. No samples were taken in 1989. Blank cells indicate no samples were taken.
Source: National Arbovirus Monitoring Program database

Chapter 3

Australia's geography, weather patterns and livestock distributions

The distribution of bluetongue, Akabane and ephemeral fever viruses in Australia is determined by complex interactions of geography, climate, the host, the vectors and the viruses. These interactions result in the southern and inland areas of Australia being continuously free of livestock arboviruses, while the northern and some of the eastern coastal areas have a seasonal activity that can vary markedly from year to year. This section summarises the principal factors causing the observed patterns of arbovirus distribution in Australia and discusses the nature of the interactions between them.

3.1 Geography

Australia (Figure 3.1) is an island continent with a land area of around 7 692 030 square kilometres. In area, Australia is almost as large as the United States of America (excluding Alaska), about 50% larger than Europe (excluding the former USSR) and 32 times larger than the United Kingdom.

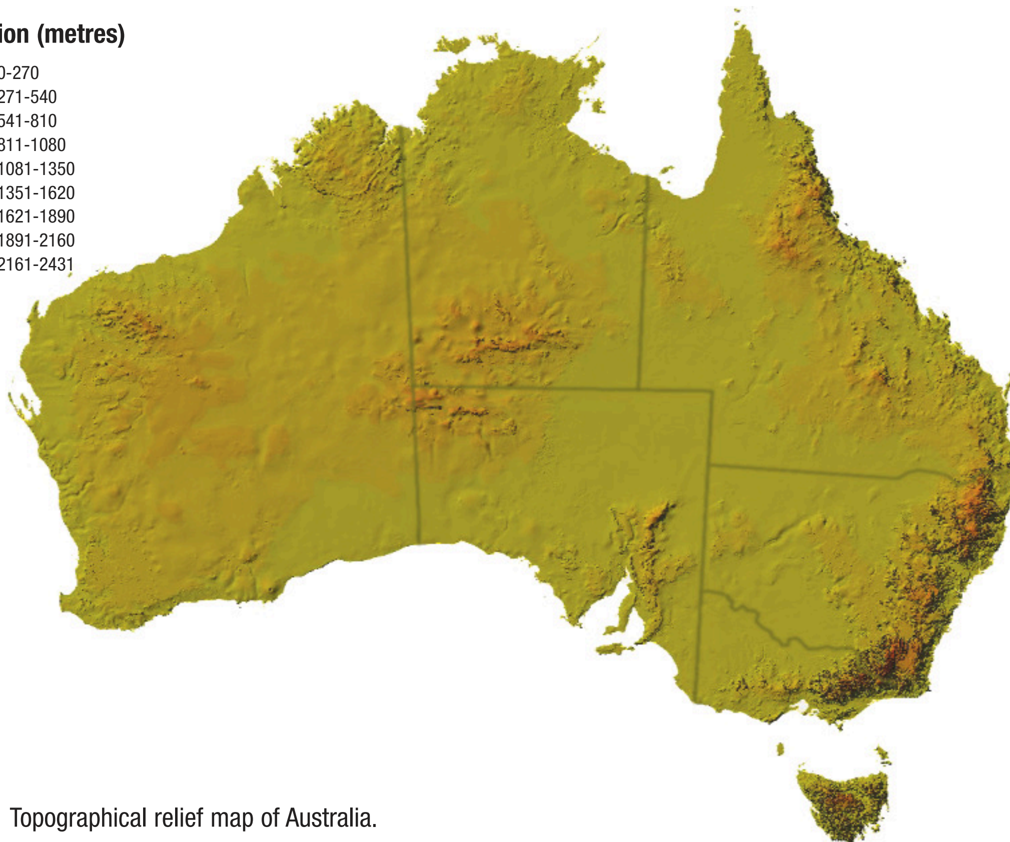

Figure 3.1 Topographical relief map of Australia.

Australia is separated from its northern neighbours by the Torres Strait and the Timor, Arafura and Coral Seas. Immediately to the north of the country lies the island of Papua New Guinea. At its closest point, Papua New Guinea is about 120 kilometres from mainland Australia.

Australia is the lowest, flattest and, apart from Antarctica, driest of the continents. The age of landforms in Australia is generally measured in many millions of years, in contrast to Europe and North America, where some landscapes date back only to the retreat of great ice sheets some 20 000 years ago. This gives Australia a distinctive physical geography, illustrated in Figure 3.1.

The Australian terrain consists mainly of low plateaux, with a number of deserts found in the vast, dry interior, but there are fertile plains in the southeast. Land use is varied, with 6% being arable, 58% pastures, 14% forest and woodland and 22% other uses.

Geographical features of greatest relevance to the distribution of livestock arboviruses are the Great Dividing Range (New South Wales, Queensland) and the Great Sandy Desert (Western Australia). These features divide zones of possible livestock arbovirus activity and zones continuously free of activity. The Great Dividing Range extends along most of the eastern seaboard of the continent and is particularly significant because it separates the dry interior from the moist east and southeast coasts. For example, the mean annual rainfall at Coffs Harbour, on the New South Wales coast, is 1647 mm, whereas the mean annual rainfall of Moree, an inland centre at approximately the same latitude and only 330 km away, is 578.6 mm. The Great Sandy Desert in the north and centre of Western Australia is bounded by the Kimberley to the north, the Pilbara Desert to the southwest and the Gibson Desert to the south. This extensive desert separates a region of high rainfall to the north that is suitable for arbovirus activity from one of low rainfall to the south that is continuously free of livestock arboviruses. For example, Derby, located on the coast of northern Western Australia, has a mean annual rainfall of 709.9 mm, compared with 327.1 mm for Port Hedland, southwest of the Great Sandy Desert.

3.2 Climate

Australia features a wide range of climatic zones, from the tropical regions of the north, through the arid expanses of the interior, to the temperate regions of the south. The landmass is relatively arid, median rainfall being less than 600 mm per year in 80% of the country and less than 300 mm in 50% of the country, with an average of 450 mm. Seasonal fluctuations can be large, with temperatures ranging from above 50°C to well below zero.

Detailed statistics on the climate throughout Australia are available from the Commonwealth Bureau of Meteorology website.[4] The information available includes monthly and quarterly rainfall and temperature maps of Australia.

Although the climate can be described as predominantly continental, the insular nature of the landmass modifies the general continental pattern. Australia experiences many of nature's more extreme phenomena, particularly droughts, floods, tropical cyclones, severe storms and bushfires.

The generally low relief of Australia causes little obstruction to the atmospheric systems controlling the climate. A notable exception is the Great Dividing Range. From May to October, anticyclones or high-pressure systems

4 www.bom.gov.au

pass from west to east across the continent and may remain almost stationary over the interior for several days. These anticyclones may be up to 4000 km wide and rotate anticlockwise. Northern Australia is thus influenced by mild, dry southeast winds, while southern Australia experiences cool, moist westerly winds. The westerlies and the frontal systems associated with extensive depressions (lows or extra-tropical cyclones) travelling over the Southern Ocean control the climate of southern Australia during the winter season, causing rainy periods. Periodic northwest cloud bands in the upper levels of the atmosphere over the continent may interact with southern systems to produce rainfall, particularly over eastern areas. Cold outbreaks occur, particularly in southeast Australia, when cold air from the Southern Ocean is directed northwards by intense depressions of up to 2000 km in diameter. Cold fronts associated with southern depressions or with secondary depressions over the Tasman Sea may produce strong winds and large day-to-day variations in temperature in southern areas, particularly on the southeast coast.

From November to April, the anticyclones travel from west to east on a more southerly track across the southern fringes of Australia, directing easterly winds over the continent. Fine, warmer weather predominates in southern Australia with the passage of each anticyclone. Heat waves occur when there is an interruption to the eastward progression of the anticyclone ('blocking') and winds back northerly, then northwesterly. Northern Australia comes under the influence of summer disturbances associated with the southward intrusion of warm moist monsoonal air from north of the intertropical convergence zone, resulting in a hot rainy season. Southward dips of the monsoonal low-pressure trough sometimes spawn tropical depressions and may prolong rainy conditions over northern Australia for up to three weeks at a time.

The climate of eastern and northern Australia is influenced by the southern oscillation, a seesawing of atmospheric pressure between the northern Australian–Indonesian region and the central Pacific Ocean. The oscillation is one of the most important causes of climatic variation over eastern and northern Australia. Its strength is defined by the southern oscillation index, which is a measure of the difference in atmospheric pressure at sea level between Tahiti in the central Pacific and Darwin in northern Australia. At one extreme of the oscillation, the pressure is abnormally high at Darwin and abnormally low at Tahiti. These conditions are usually accompanied by severe and widespread drought over eastern and northern Australia. They commence early in the year, last for about 12 months and recur every two to seven years. In general, they are immediately preceded or followed by the opposite extreme, with abnormally low pressures at Darwin, abnormally high pressures at Tahiti and above-average rainfall over eastern and northern Australia. Extreme dry years are referred to as El Niño ('baby boy' in Spanish) and extreme wet years as La Niña ('baby girl').

Annual rainfall

Figure 3.2 shows average annual rainfall over the Australian continent. The lowest rainfall is in the vicinity of Lake Eyre in South Australia, where the median annual rainfall is around 100 mm. Another very low rainfall area is in Western Australia, in the region of the Giles–Warburton Range, which has a median annual rainfall of about 150 mm. A vast region, extending from the west coast near Shark Bay across the interior to southwest Queensland and northwest New South Wales, has a median annual rainfall of less than 200 mm. This region is not normally exposed to moist air masses for extended periods and rainfall is irregular, averaging one or two days per month. However, in favourable synoptic situations, which occasionally occur over extensive parts of the region, up to 400 mm of rain may fall within a few days, causing widespread flooding.

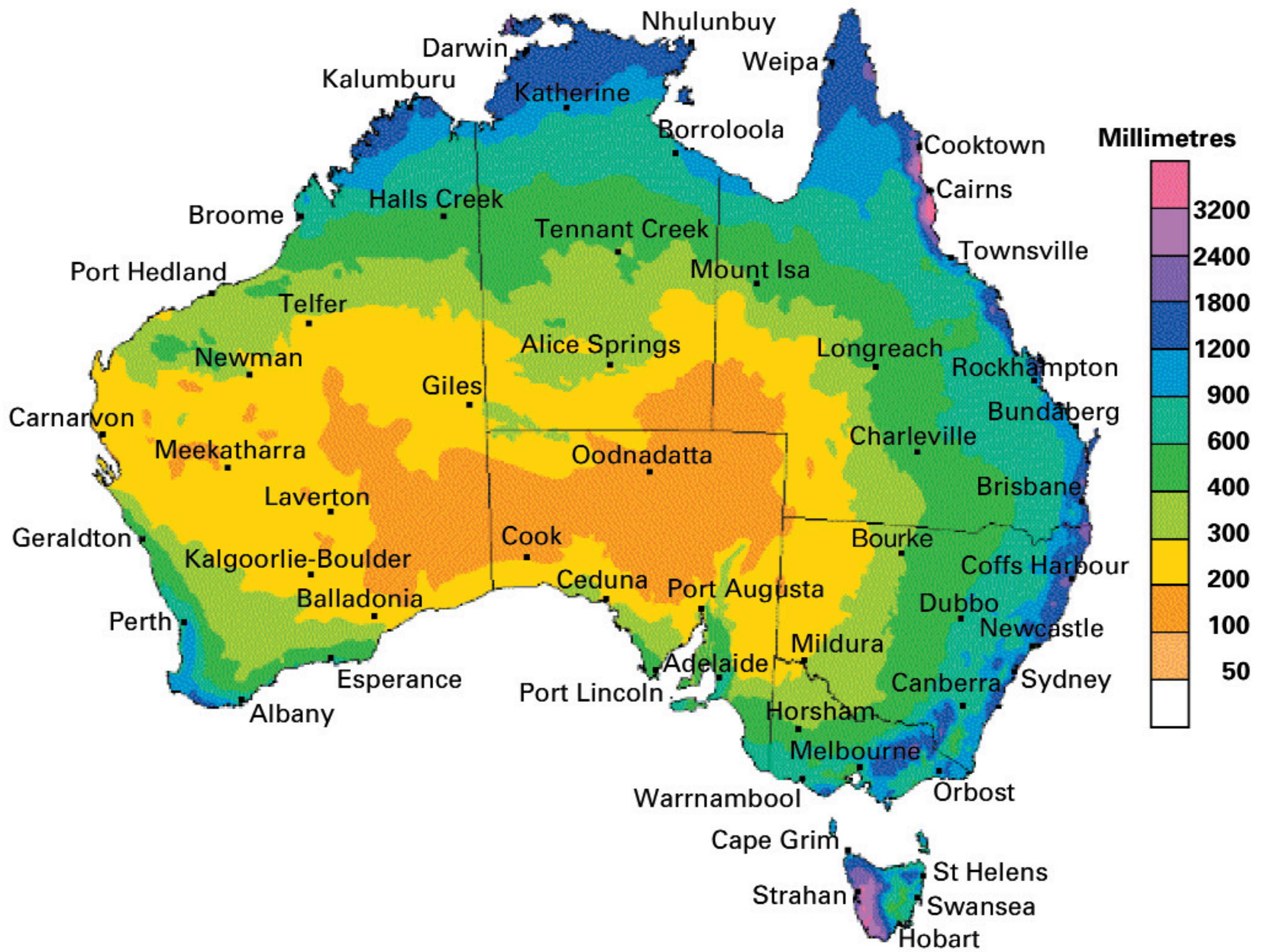

Figure 3.2 Average annual rainfall (based on a standard 30-year climatology 1961–90).
Source: Australian Bureau of Meteorology

The region with the highest median annual rainfall is the east coast of Queensland, between Cairns and Cardwell: in Tully, the median rainfall was 4048 mm in the period 1924–87. The mountainous region of western Tasmania also has a high annual rainfall, with Lake Margaret having a median of 3565 mm in the period 1911–87. In the mountainous areas of northeast Victoria and some parts of the east coastal slopes, there are small pockets with median annual rainfall greater than 2500 mm. The Snowy Mountains area in New South Wales also has a particularly high rainfall; the highest median annual rainfall for this region is 3200 mm. Small areas on the western slopes above 2000 metres elevation probably have a median annual rainfall approaching 4000 mm.

Seasonal rainfall

The rainfall pattern of Australia is strongly seasonal in character, with a winter regime in the south and a summer regime in the north. Figure 3.3 shows a series of monthly median rainfall maps of Australia based on long-term averages. The dominance of rainfall over other climatic elements in determining the growth of specific plants in Australia has led to the development of a climatic classification based on two main parameters — median annual rainfall and the incidence of seasonal rainfall. Evaporation and the concept of rainfall effectiveness are taken into account to some extent in this classification by assigning higher median annual rainfall limits to the summer zones than to the corresponding uniform and winter zones. The main features of the seasonal rainfall are:

- the marked wet summer (the 'monsoon') and dry winter of northern Australia;
- the wet summer and relatively dry winter of southeastern Queensland and northeastern New South Wales;
- uniform rainfall in southeastern Australia, including much of New South Wales, parts of eastern Victoria and southern Tasmania;
- the marked wet winter and dry summer of southwest Western Australia and, to a lesser extent, much of the remainder of southern Australia directly influenced by westerly circulation ('Mediterranean' climate); and
- an arid area comprising about half the continent extending from the northwest coast of Western Australia across the interior and reaching the south coast at the head of the Great Australian Bight.

Temperature

Figure 3.4 shows the distinctly seasonal nature of temperature variations. The seasonal rainfall and temperature maps (Figures 3.3 and 3.4) together illustrate the monsoonal patterns of rain in the north and the seasonally low temperatures in the south and along the east coast.

3.3 Factors affecting host populations

Australia is a significant producer and a major exporter of livestock, livestock products and livestock genetic material. Animal production in Australia is largely based on extensive grazing and is dominated by wool, sheep meat, beef and dairy production, with smaller intensive pig and poultry industries. The livestock industries extend from the beef cattle areas of tropical north Queensland to the sheep areas of southern Tasmania, and from the dairying areas of coastal New South Wales to the merino wool-producing areas of Western Australia.

Beef cattle

Cattle are raised over much of Australia, although geographical and climatic features such as steep mountains or deserts render certain areas unsuitable for cattle. There are two main cattle production systems in Australia. Across the north of the country, cattle are produced on extensive holdings, grazing native pastures at low stocking rates. Tropical breeds, better adapted to the harsh conditions in the north, dominate. The main outputs are beef, animals for lot feeding and live cattle exports. In southern Australia, cattle are produced on smaller holdings and graze mainly on improved pastures. Temperate breeds, either British or continental-derived, dominate. Smaller and younger animals are produced, mainly for the Australian domestic market.

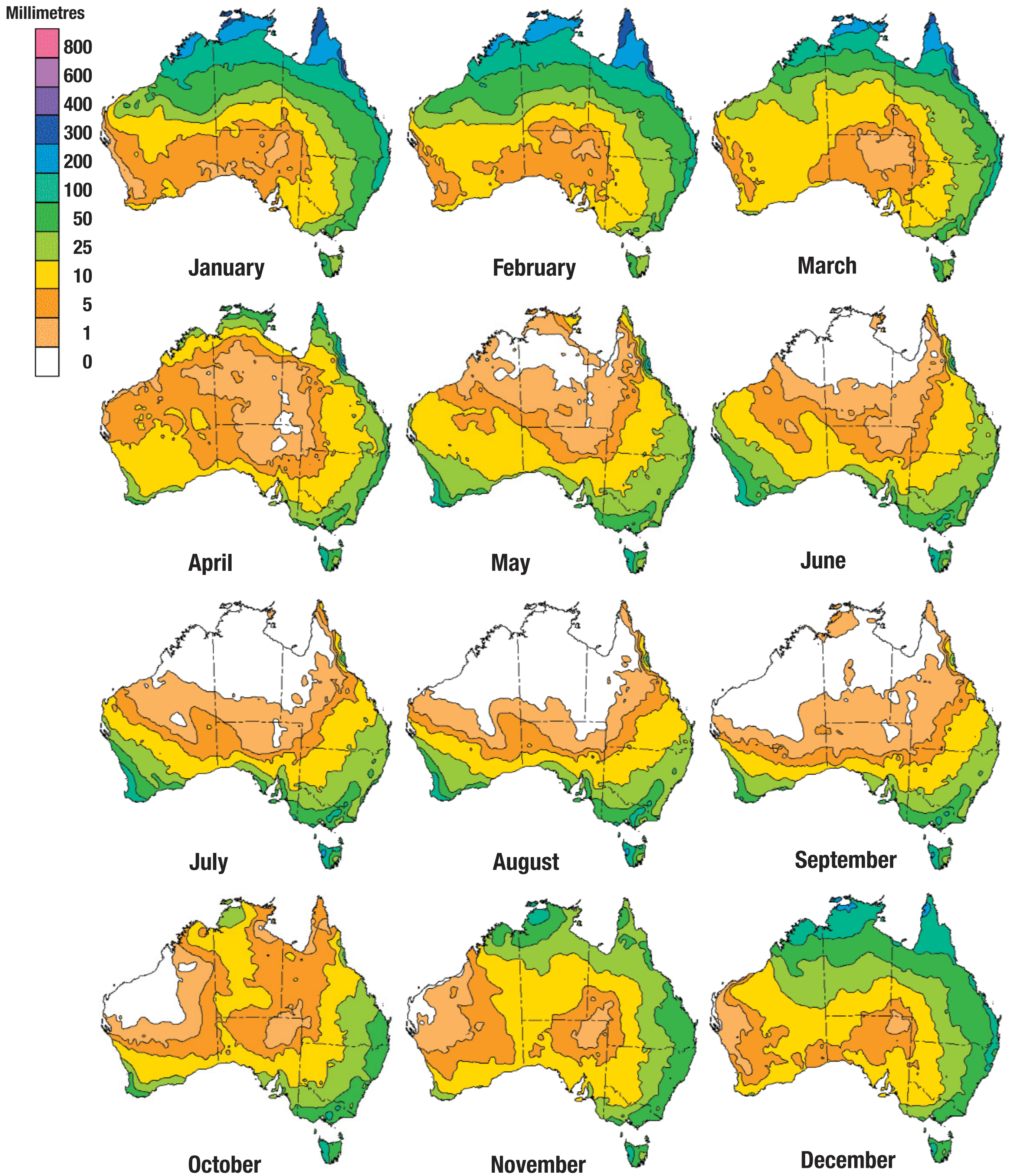

Figure 3.3 Long-term monthly median rainfall, January–December.
Source: Australian Bureau of Meteorology 1996.

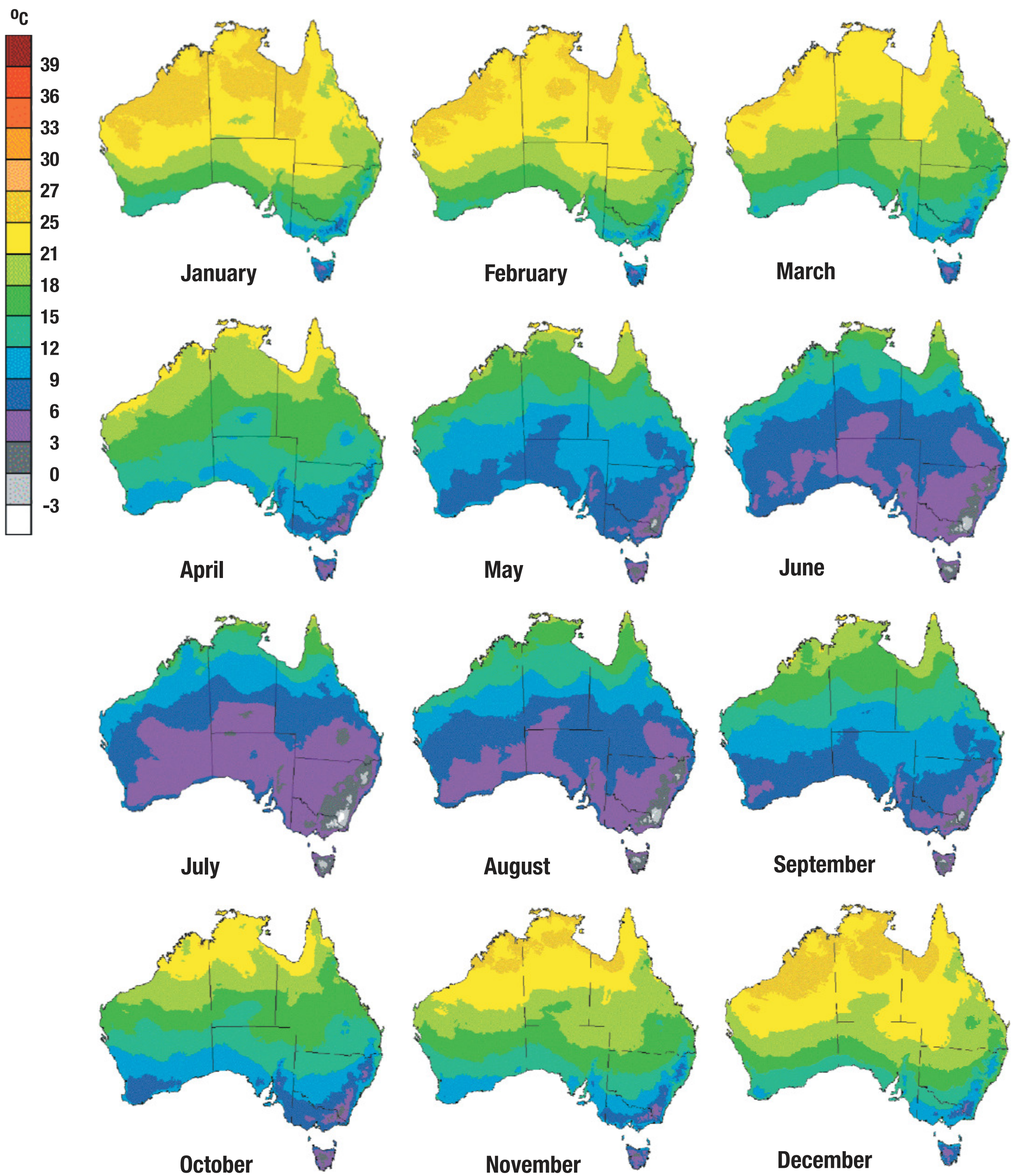

Figure 3.4 Average monthly minimum temperature, January–December, 1961–90.
Source: Australian Bureau of Meteorology.

Although pasture feeding is the major method of beef production, the use of feedlots to finish cattle for specific markets has been a significant recent development, with feedlot capacity doubling over the past five years. Most of the feedlots are located in the wheat–sheep zone of New South Wales and southern Queensland. The growth of the live cattle export trade has been another important feature of the Australian beef cattle industry in recent years, providing an alternative to slaughter for producers in northern Australia. Most of the exported cattle go to the Middle East or to Southeast Asian countries.

Dairy cattle

The Australian dairy industry operates in all States and is the third largest rural industry in terms of wholesale production value. Over the past 20 years, the dairy industry has been restructured extensively as a result of changes in government regulation of the industry and in domestic and export markets for dairy products. The number of dairy farms has more than halved and cow numbers have fallen by 20%. However, production per cow and overall production efficiency have increased markedly over the same period. The average herd comprises 120 milking cows, with a trend toward larger herds. Most dairy farms are family owned and operated.

Sheep

Sheep are produced over a wider range of conditions, from the arid and semiarid inland to the higher rainfall areas of southeastern Australia. The southwest corner of Western Australia is also an important sheep-producing area. Most sheep in Australia are produced as part of a mixed farming enterprise, often for lamb production and frequently with crops, beef and dairy cattle. Sheep numbers fluctuate according to seasonal conditions, movements in wool prices and the relative profitability of other enterprises. Numbers have been declining slowly since a maximum of 173 million head was reached in 1990. Australia is the world's largest supplier of apparel wool. The sheep meat industry developed alongside the wool industry but is now an important industry in its own right. Wool and mutton production is generally confined to higher rainfall areas with good pasture conditions.

Tables 3.1, 3.2 and 3.3 show the historical cattle and sheep population of Australia.

Figures 3.5 and 3.6 show the distributions of cattle and sheep. Figure 3.7 shows the area of Australia in which there is a high density of cattle and a low density of sheep. This area corresponds approximately to the distribution of bluetongue and Akabane viruses, although some large populations of cattle are present in the south and west of Australia where the viruses do not appear.

Table 3.1 Cattle and sheep population (thousands) of Australia

Year	Cattle	Lambs and sheep
1861	3 958	20 135
1871	4 276	41 594
1881	7 527	62 184
1891	10 300	97 881
1901	8 640	70 603
1911	11 745	98 066
1921	13 500	81 796
1931	11 721	110 568
1941	13 256	122 694
1951	15 229	115 596
1961	17 332	152 579
1971	24 373	177 792
1981	25 168	134 407
1991	23 662[a]	163 238
1992	23 880[a]	148 203
1993	24 062[a]	138 099
1994	25 758[a]	132 569
1995	25 731[a]	120 862
1996	26 377[a]	121 116
1997	26 780[a]	120 228
1998	26 826[a]	117 494
1999	26 578	115 500

[a] Excluding house cows and heifers
Source: Australian Bureau of Statistics figures for 1997–98

Table 3.2 Cattle and calves (thousands) by State/Territory[a]

Year	NSW	Vic	Qld	SA	WA	Tas	NT	Aust[b]
1993	5783	3689	9873	1104	1648	605	1347	24 062
1994	6515	4189	9942	1202	1806	679	1435	25 758
1995	6236	4280	9974	1216	1899	693	1421	25 731
1996	6390	4396	10 214	1219	1924	718	1503	26 377
1997	6511	4411	10 422	1181	1909	725	1609	26 780
1998	6351	4142	10 867	1214	1973	728	1567	26 851
1999	6291	4125	10 748	1183	1931	724	1567	26 578

[a] Excluding house cows and heifers
[b] Includes the Australian Capital Territory
Source: Australian Bureau of Agricultural and Resource Economics, *Australian Commodity Statistics 2000*

Table 3.3 Sheep and lambs (millions) by State/Territory

Year	NSW	Vic	Qld	SA	WA	Tas	NT	Australia
1993	48.1	23.6	13.4	15.7	33.0	4.3		138.1
1994	46.5	23.4	11.5	14.7	32.0	4.3		132.6
1995	40.5	21.4	11.6	13.2	30.2	3.9		120.9
1996	41.1	22.0	10.7	13.6	29.8	3.9		121.1
1997	42.4	22.3	10.5	13.1	27.8	4.0		120.2
1998	40.8	21.1	11.0	13.1	27.5	3.9		117.5
1999	40.6	20.9	10.6	13.1	28.3	3.8		115.5

Source: Australian Bureau of Agricultural and Resource Economics, *Australian Commodity Statistics 2000*

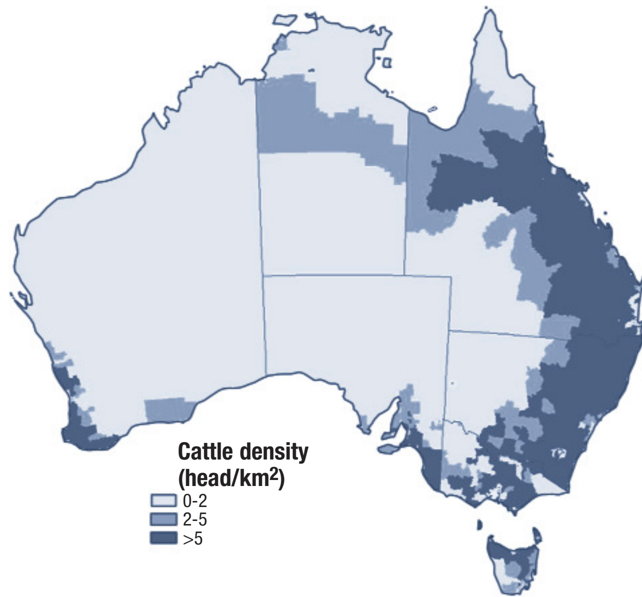

Figure 3.5 Distribution of cattle in Australia, showing areas of high, medium and low density.
Source: Australian Bureau of Statistics, 1996.

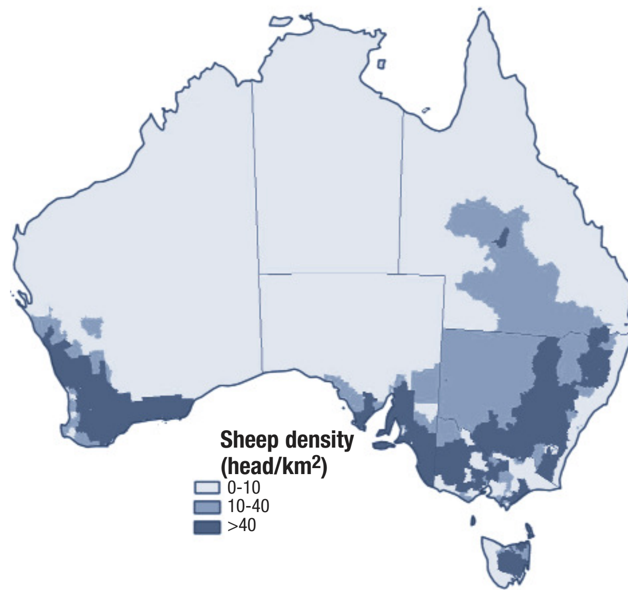

Figure 3.6 Distribution of sheep in Australia, showing areas of high, medium and low density. The ranges represent approximately equivalent stocking rates to those in Figure 3.5.
Source: Australian Bureau of Statistics, 1996.

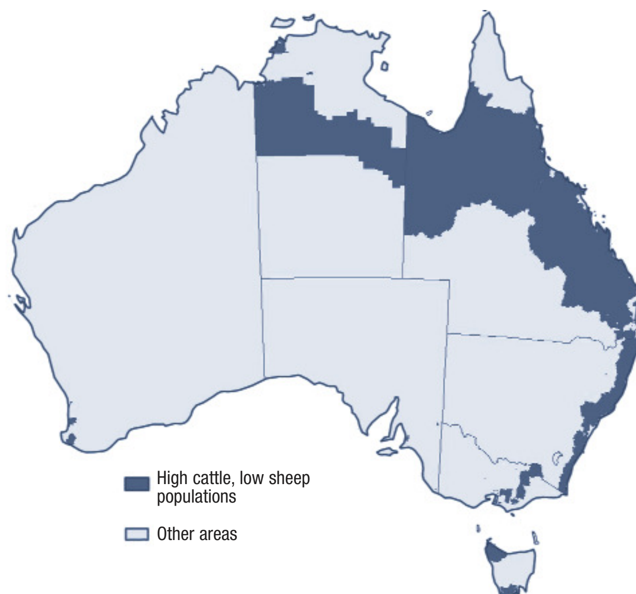

Figure 3.7 Area of Australia with high cattle population and low sheep population.
Source: Australian Bureau of Statistics, 1996.

3.4 Factors affecting vector distribution

The distribution of an arbovirus within a vertebrate host population is determined by the distribution of its insect vector. In turn, there are many geographical and environmental factors influencing the survival, spread and ultimate distribution of a vector.

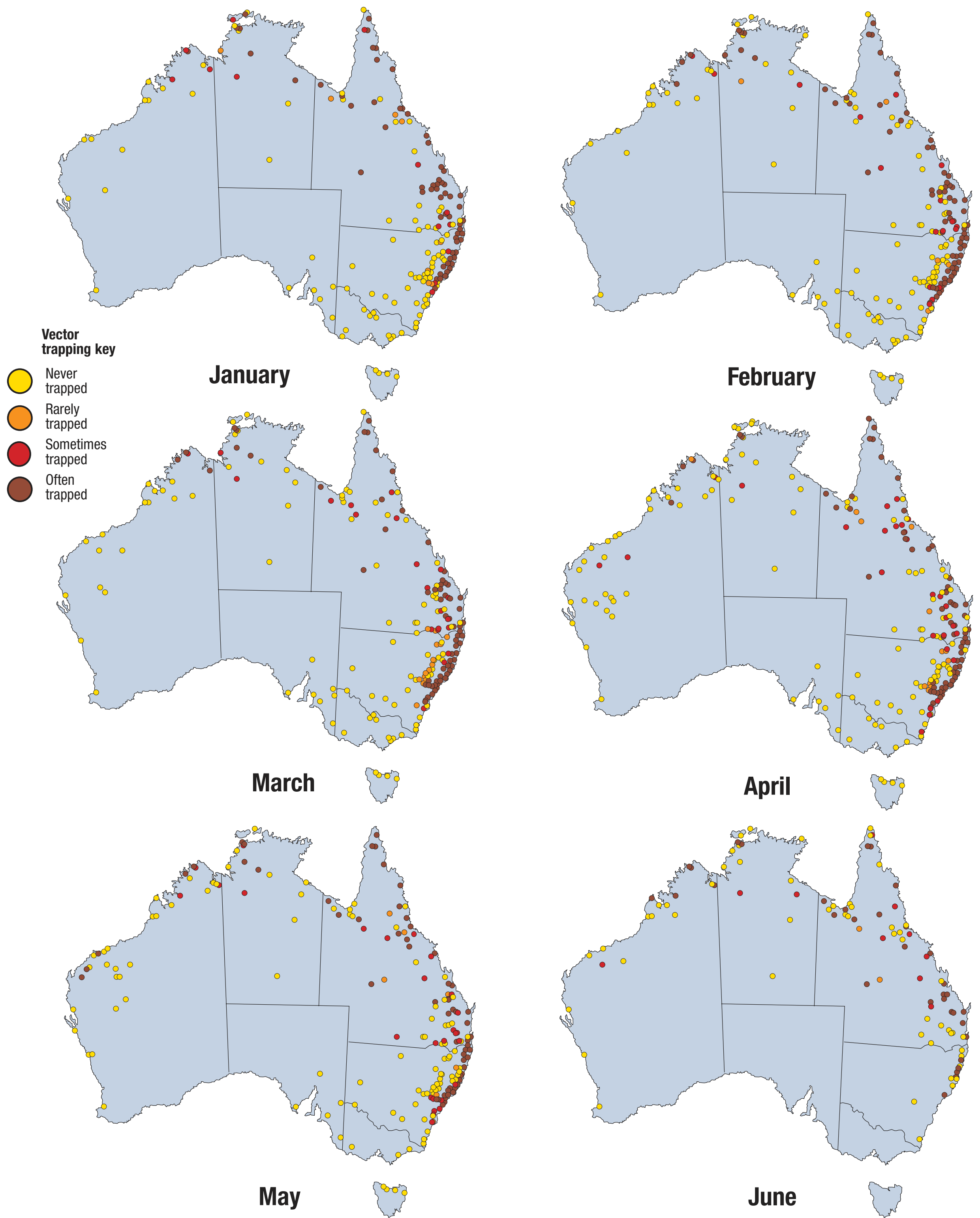

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Figure 3.8 Long-term monthly distribution of *Culicoides brevitarsis*, January to December, 1970–99. Spots represent vector trapping sites; colours indicate the number of *C. brevitarsis* identifications at that site in each month (see key).
Source: NAMP national database.

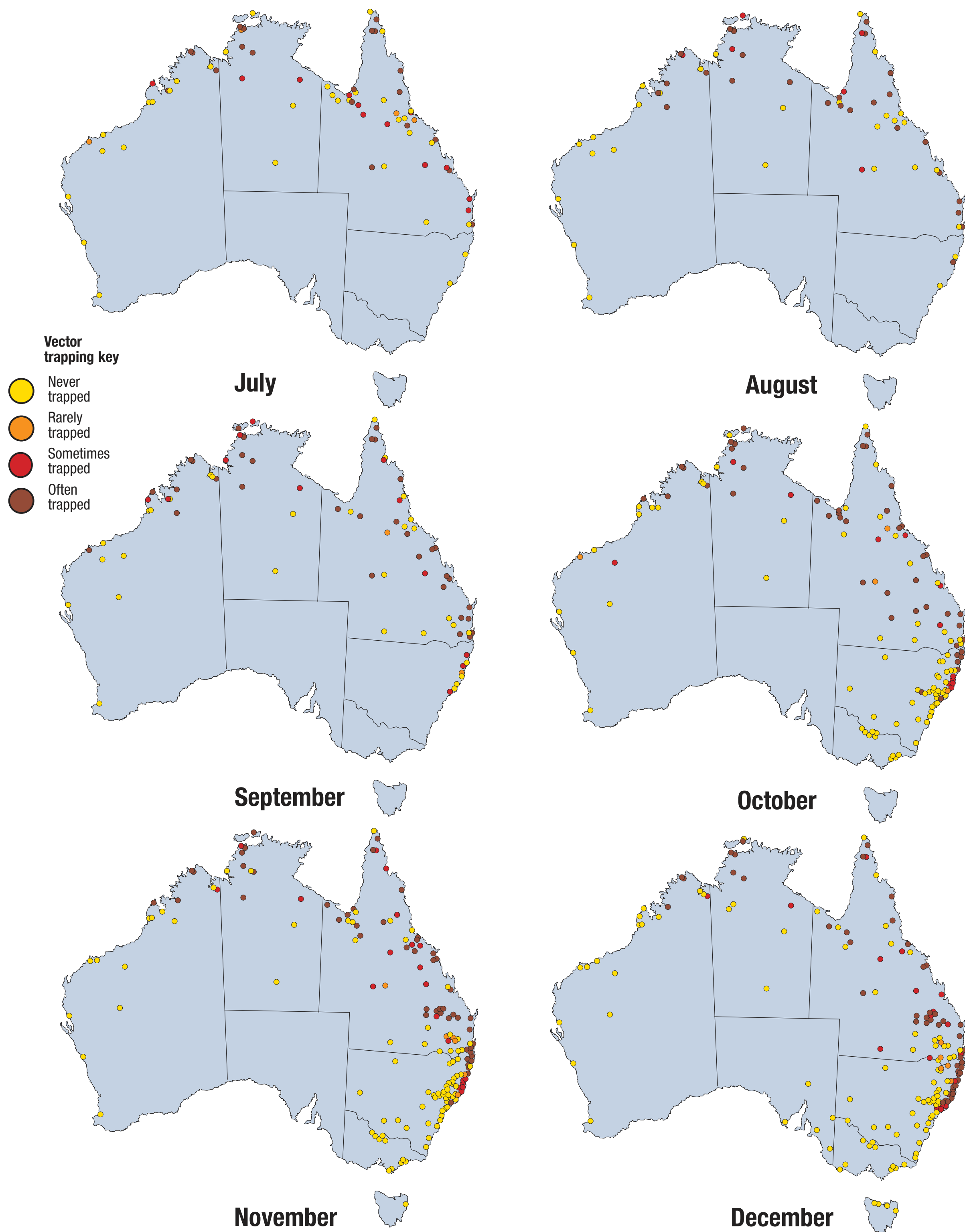


Figure 3.8 Continued

A biting midge, *Culicoides brevitarsis,* is the principal vector of bluetongue and Akabane in Australia. The vector's main hosts, members of the bovine species, play a significant role in its distribution, because this biting midge can only breed in bovid dung. Environmental factors also affect distribution of the vector. High and steep mountains can form natural barriers to the movement of *Culicoides.* Very low temperatures also limit distribution: *C. brevitarsis* cannot survive below about 7°C. Figure 3.8 shows the seasonal variation in the distribution of *C. brevitarsis* based on vector trapping data since 1970. When compared with the monthly minimum temperature maps (Figure 3.4), the influence of temperature on vector distribution is clear. Low rainfall also limits the survival and/or spread of *C. brevitarsis*. The midge cannot survive in areas with an annual rainfall below 400 mm, such as the vast desert area of inland Australia, and may be temporarily eliminated from a region by drought conditions.

In regions where there are distinct seasons, midges are absent for many months each year, especially in winter. Once the midge population is lost from such an area, it may require several 'favourable' years before the population can re-establish. The existence of suitable climatic conditions for a short time does not usually lead to a midge population becoming established unless the area is immediately adjacent to a vector region. In this case, a temporary population may become established until harsh conditions return, but there are rarely sufficient midges to support virus transmission. Very high temperatures and rainfall can reduce midge populations, but they usually have less impact than low temperatures and rainfall.

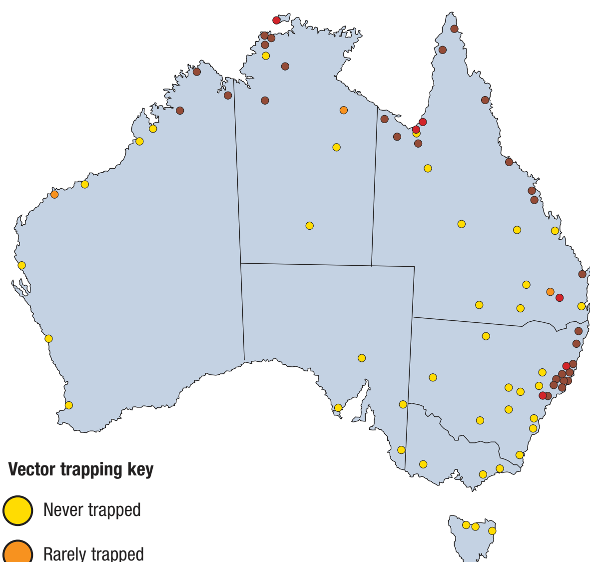
Even when climatic conditions are suitable, if livestock, especially cattle, are excluded from a large area due to extreme features of the terrain, or for conservation reasons, there will be no bovid dung for *C. brevitarsis* to breed in and no hosts for midges to feed on.

In Australia, the complex interaction of geography, climate and cattle distribution confines *C. brevitarsis* to the northern parts of Western Australia and the Northern Territory, the northern and east coastal regions of Queensland and the narrow coastal strip of the northern half of New South Wales. There are no significant sheep populations in these areas (Figure 3.6) but the cattle density can be high (Figure 3.5). As the climatic conditions vary, vector population densities will also vary. Even in an area where vectors are continually present, the midge population may be too small, or cattle too dispersed, to support virus transmission.

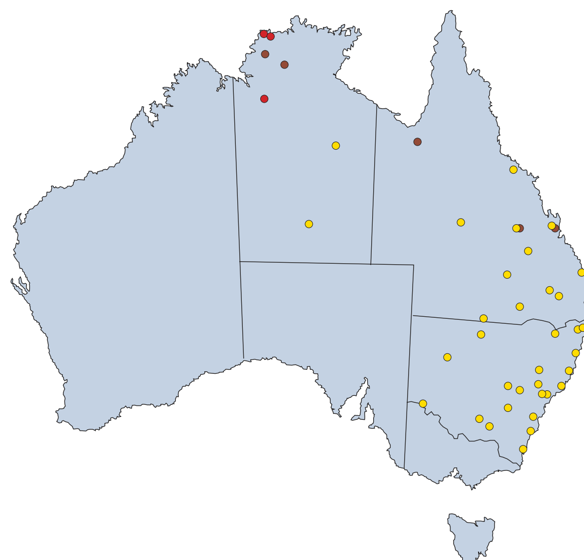
The presence of *C. brevitarsis* in a region, even in large numbers, does not imply that arboviruses will be present. For example, in the summer and autumn of 1996, New South Wales had a normal distribution and population density of *C. brevitarsis* (Figure 3.9a), yet no transmission of bluetongue or Akabane virus was detected in sentinel cattle anywhere in the State (Figures 3.9b and 3.9c). In other years, with populations of similar density, sentinel cattle in the vector region can be infected with Akabane virus, bluetongue virus, or both. The reasons for the difference in transmission between years are not clear. Seroconversions to these viruses beyond the limits of *C. brevitarsis* (Sections 5.1 and 5.2) have not been recorded.

The main vector of bovine ephemeral fever virus is believed to be the mosquito *Culex annulirostris*, which is less susceptible to climatic extremes than *Culicoides brevitarsis*. While this virus is found mainly in regions where *C. brevitarsis* is present, it is occasionally found in more remote locations, as occurred in 1996 (Figure 3.9d).

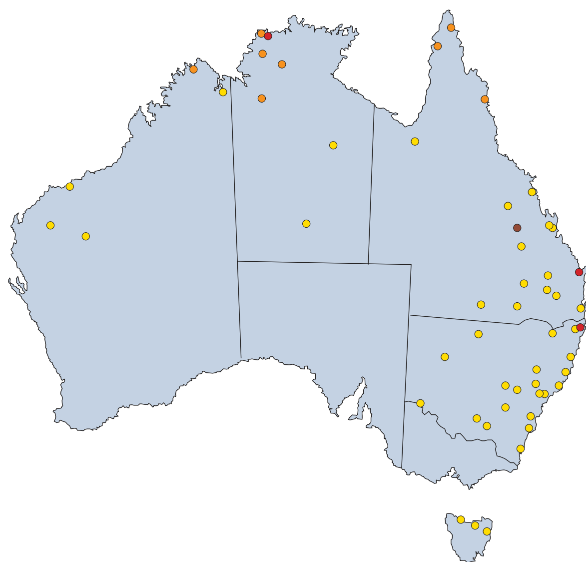
(a) *Culicoides brevitarsis*



(c) *Akabane virus*



(b) *Bluetongue virus*



(d) *Ephemeral fever virus*

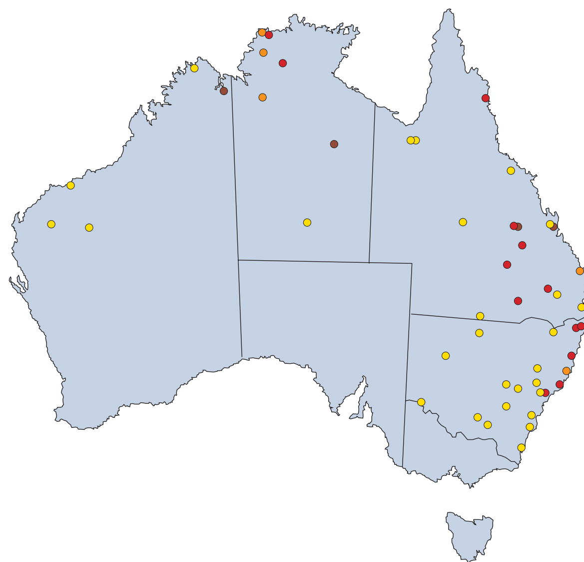


Figure 3.9 Distribution of (a) *Culicoides brevitarsis*, and of (b) bluetongue, (c) Akabane and (d) ephemeral fever virus seroconversions, as detected by NAMP vector trapping sites and sentinel herds, 1995–96.
Source: NAMP national database.

Chapter 4

Insect vectors

4.1 Introduction

A virus that spreads from animal to animal with a multiplication stage in an insect has evolved in a specific virus–insect–animal relationship. When the virus multiplies in the animal, it must at some stage circulate in the bloodstream to be available to the blood-feeding insects that are adapted to feed on that animal. The virus must be capable of infecting an insect that imbibes it and of subsequently being transmitted in the saliva to a fresh vertebrate host.

Insect vectors are subject to significant and continuous attrition by predators, weather conditions and viral and bacterial diseases. A very small proportion survive from the egg, through the larval stages and intervals between successive bloodmeals, to reach the stage of transmitting a particular virus.

The efficiency of virus transmission depends on a combination of factors: efficiency of growth within an insect, vector population numbers and availability of susceptible animals. With a few exceptions, arboviruses, including those that cause disease in animals, are harmless to the insect vector.

An understanding of the biology and interactions of the virus, insect vector and animal host is required to develop surveillance and control systems. New risks may emerge if established ecologies are disturbed by human intervention.

Taxonomic studies in Australia over the last 40 years have defined the insect species significant for arbovirus transmission. Particular species of biting midge, *Culicoides*, spread bluetongue and Akabane virus, while certain *Culex* and *Anopheles* mosquitoes transmit bovine ephemeral fever (BEF).

4.2 Biting midges (*Culicoides*)

Significant species for arbovirus transmission

In Australia, 123 species of biting midge of the genus *Culicoides* have been formally described, with a further 150 species still to be described. Most of these species feed on indigenous birds or marsupials and are thus irrelevant to virus transmission between domestic animals. A few species (eg *C. marksi*, *C. dyce*i and *C. victoriae*) feed on cattle, sheep and horses as well as indigenous mammals. However, experimental and epidemiological studies have shown that these indigenous species do not spread bluetongue or Akabane viruses.

C. brevitarsis, *C. wadai, C. fulvus* and *C. actoni* support the multiplication of bluetongue viruses (Table 4.1). Two of these species, *C. brevitarsis* and *C. wadai*, depend on cattle or buffalo dung for breeding sites, and only became established in Australia some years after the introduction of domesticated livestock to northern areas in 1825. Indirect evidence suggests that the major southern spread of *C. brevitarsis* through southern Queensland into New South Wales occurred between the 1870s and 1931.

Table 4.1 Species of *Culicoides* associated with bluetongue (BLU) or Akabane (AKA) virus in Australia

***Culicoides* species**	**Breeding site**	**Vertebrate hosts**	**Recent introduction**	**Virus isolated from wild insect**	**Vector status[a]**	
					Bluetongue	**Akabane**
C. brevitarsis	Cattle dung, buffalo dung	Cattle, sheep, horses	Yes	BLU, AKA	2	Highly probable
C. wadai	Cattle dung	Cattle, sheep, horses	Yes	BLU, AKA	1	Unknown
C. fulvus	Tropical subcoastal (larval habitat unknown)	Cattle, buffalo	No	BLU	2	Unknown
C. actoni	Rotting native fruit	Cattle, marsupials	No	BLU	2	Unknown

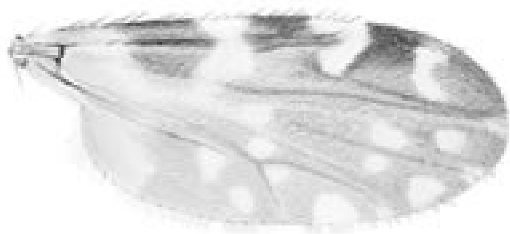
[a] vector status:
1 = infected with laboratory-adapted virus but transmission not proven
2 = isolated from wild-caught insects and transmitted from vertebrate to *Culicoides* to vertebrate

Identification of species

Culicoides species are very small insects, visible to the naked eye, with a wing length of about 0.9 mm. The most useful visual characteristic to separate species is the pattern of markings on the wings (see Figure 4.1).

C. wadai

C. victoriae

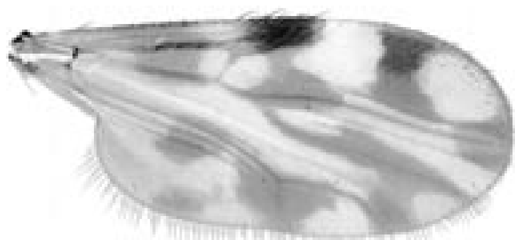


C. peregrinus

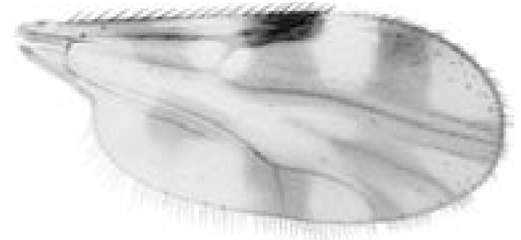
C. oxystoma

C. marksi

C. fulvus



C. brevitarsis



C. actoni

Figure 4.1 Wing patterns used to identify eight species of *Culicoides*.
Source: Dr AL Dyce.

Life cycles

Culicoides that spread Akabane and bluetongue viruses lay eggs that hatch about one day after being deposited in cattle or buffalo dung. Under ideal conditions, larvae go through three stages before pupating and emerging as adults in 7–10 days. Females start to mate and search for a bloodmeal on the day they emerge from dung. The time spent searching for a bloodmeal varies between species and is limited by temperature and humidity. *Culicoides* are attracted to hosts by stimuli such as exhaled carbon dioxide, shape and body heat, and are particularly active at twilight and just before sunrise. Strong winds can disperse *Culicoides* over long distances.

After the first bloodmeal, females mature and lay a batch of eggs in fresh dung (usually less than seven days old). Though midges may be ready for another bloodmeal in 1–2 days, it requires 1–2 weeks (depending partly on the temperature) after an infective bloodmeal for virus to have infected the midge and be excreted in the saliva during subsequent blood feeds.

Culicoides species can survive winter by slow, continuous development when conditions are suitable. *C. brevitarsis* can survive mild winter temperatures for short periods in larval or pupal stages in dung. However, larvae of *C. brevitarsis* cannot survive if the temperature remains below 17°C for 50 days (most regions of New South Wales) or if the dung is too hot (eg in inland areas of northern Queensland, where soil surface temperatures can exceed 50°C in summer). In such areas, small populations exist through the winter. Sentinel herd serology has shown that Akabane and other viruses may spread in the late winter instead of the summer in these areas.

None of the Australian *Culicoides* species has been successfully colonised for laboratory studies. However, in areas where the dung-breeding species are active, dung can be collected and kept in a temperature-controlled room until the adult *Culicoides* emerge. This technique is used to produce adults for infectivity and biology studies, to link larval stages to adults or to assess the mortality caused by residues of insecticides administered systemically to cattle. In Australia, Dr AL Dyce has developed methods to age *Culicoides* species and determine how many batches of eggs they had matured in.

Distribution and dispersal

All the *Culicoides* species that can carry bluetongue or Akabane viruses in Australia are also found in Indonesia, but only some of these species occur in Papua New Guinea. Additional species able to transmit bluetongue virus are found in Indonesia and should be detected by the current surveillance system if they enter Australia.

The distribution of the four *Culicoides* species that transmit bluetongue or Akabane viruses in Australia is influenced by climate. During the active season, extension from the regions where these species can persist all year occurs in the direction of low-level prevailing winds. Alternatively, violent winds, very heavy rains and floods can severely reduce numbers of adults in known vector areas and can wash out breeding sites, while drought may contract the vector range. The extent of the seasonal expansion of range depends on a number of factors and has only been well studied in New South Wales. The southward extension of *Culicoides* into Australia has reached relatively stable limits, which vary from species to species. *C. brevitarsis* is more widely distributed than *C. wadai*, *C. fulvus* and *C. actoni*, which are limited to the far north and the northeastern coastal strip.

C. wadai was first identified in the Northern Territory in 1971 but has extended its range down the east coast of Queensland and into northern New South Wales. It was found near Brisbane in 1983 and in Lismore and Grafton, on the northern New South Wales coast, in 1984 and 1985 respectively. An extensive survey between 1989 and 1992 failed to detect *C. wadai* in New South Wales and its distribution was thought to have contracted to southern Queensland due to several years of low summer rainfall. *C. wadai* again reached Lismore and Casino in 1998 and had extended to Grafton and Coffs Harbour by 1999. The eventual coastal limit of *C. wadai* is expected to be around the Hunter Valley of New South Wales.

C. brevitarsis is the most significant vector of Akabane and bluetongue viruses that extends south and inland. The adults take bloodmeals from cattle, sheep, horses, goats, donkeys and various species of introduced deer. However, they only lay eggs in the dung of cattle or water buffalo and are therefore not found in purely sheep-breeding areas. Summer rainfall and high cattle densities, which provide abundant breeding sites, aid the development of high population densities of *C. brevitarsis*, as seen in coastal areas of Queensland and northern New South Wales. The dispersal of these insects toward the south and west, along river valleys in northern New South Wales and inland Queensland, is critical to the epidemiology of bluetongue and Akabane viruses.

Population dynamics

During the day, the adult insects shelter in pasture grass, tussocks or vegetation along the edges of dams. As light and temperature conditions become favourable, the adults leave resting sites and form swarms, with numbers peaking around sunset. The swarms, which contain large numbers of males, are probably related to mating. There are particularly large numbers of males in dawn flights, suggesting more mating activity at this time.

Swarms may form anywhere but are more frequent near livestock. They usually form above a visual marker such as a fence post or large rock. Once the decreasing light prevents visual orientation to the markers, the females move to animals to feed. Most feeding activity occurs 1–2 hours after sunset and declines as the temperature drops. Lower levels of activity occur at sunrise than at sunset. When animals are moved indoors before sunset, the attack rate is reduced by 99% compared to animals outside.

Adult *C. brevitarsis* are apparently unable to locate preferred resting sites (grass and tussocks) in the dark and therefore rest on any pasture grass at night. Re-establishment in preferred sites occurs in the first hour after sunrise, when visual orientation is again possible. In the evening, activity may begin 2–4 hours before sunset and usually peaks in the hour before sunset. The behaviour of *C. brevitarsis* is triggered by decreasing light rather than the timing of sunset or sunrise, so cloud cover in the afternoon will bring the time of activation forward.

Host preference

Culicoides orientate towards the host that provides the strongest olfactory and visual stimulus. *C. brevitarsis*, *C. wadai*, *C. fulvus* and *C. actoni* feed on cattle, sheep, goats, deer, horses and donkeys. It is difficult to carry out experiments to determine whether a cow or a sheep is more attractive to a particular species in the same area. For example, *C. brevitarsis* captured near a mixed group of cattle and sheep are more likely to have taken cattle than sheep blood because cattle skin is more accessible to biting insects. In addition, the insects emerge from cattle dung, so have a greater chance of initially encountering cattle than sheep. Pheromones may also attract

midges to hosts. For example, within a single ruminant population, entire males show a higher incidence of seroconversion to Akabane or bluetongue viruses than castrated males or breeding females.

A sentinel flock of sheep maintained in suburban Darwin, which is hot throughout the year, did not seroconvert to several viruses infecting cattle in this area, but this could reflect the low *C. brevitarsis* population. High rates of transmission of Akabane virus may occur in sheep where very dense populations of *C. brevitarsis* are found, such as the east coast of Australia. However, most sheep flocks in eastern Australia are located west of the Great Dividing Range, which is usually free of *C. brevitarsis* because the climatic conditions are unfavourable and there are too few breeding sites to maintain a resident insect population.

Control

In South Africa, cattle are kept close to sheep in bluetongue-prone areas to minimise transmission. However, the most prevalent vector in that country is *C. imicola*, which breeds in muddy areas enriched by organic matter. If this routine were practised in Australia, it would bring the breeding sites of several species of *Culicoides* capable of carrying bluetongue and Akabane viruses closer to sheep. Housing can limit exposure but this method is only economical for valuable stud stock.

Cattle injected with avermectin insecticides have sufficient circulating pesticide to kill adult *Culicoides* that feed on them for up to 30 days after treatment. The dung of these treated cattle can kill larvae up to 42 days after the injection. This procedure reduces *Culicoides* numbers locally but does not kill insects immediately or repel them, so viruses can still be transmitted as a midge takes in a fatal dose. Some insecticides kill *Culicoides* spp more rapidly but it is not known whether they repel insects or kill them quickly enough to prevent them transmitting viruses.

4.3 Mosquitoes

The mosquito fauna of Australia is not dependent on introduced domestic animals for establishment or survival. However, the ecologies of some species have been altered by European settlement in the last 200 years. For example, dams, bores and irrigation channels have produced new breeding sites for mosquitoes in areas with low rainfall.

Mosquitoes are responsible for spreading the arbovirus that causes bovine ephemeral fever (BEF). Initially, midges were thought to spread the disease, because BEF virus was isolated from a pool of mixed *Culicoides* species in Africa in 1974. However, epidemiological evidence from an ephemeral fever epidemic in 1967–68 indicated that *C. brevitarsis* was not the major vector. The disease spread from the Gulf of Carpentaria to southern Victoria in six weeks through an area later shown to be free of *C. brevitarsis*. Intensive insect collections in Victoria among clinically ill cattle found none of the *Culicoides* species that spread Akabane or bluetongue viruses.

The timing of outbreaks and geographical location of the virus provide further evidence that mosquitoes rather than midges spread ephemeral fever. BEF virus spreads between spring and autumn. Occasionally, local epidemics develop in northern Australia in the dry winter season following out-of-season heavy rain. The response time of approximately 1–2 weeks after recent rain is sufficient time to increase mosquito numbers, but not *Culicoides* numbers. In linked studies of the two viruses, BEF virus spread much further than Akabane virus in New South Wales over a particular period and BEF virus extended west and south of the limits of *C. brevitarsis*.

BEF virus has been isolated from *Anopheles bancroftii* and from a mixed pool of culicine species of mosquito caught in the wild. *Culex annulirostris*, the species whose southern limits most closely fit those of the southernmost spread of ephemeral fever, is probably the most important vector for ephemeral fever in Australia. Although BEF virus has not been isolated from *Cx. annulirostris*, when it is fed to these insects in bloodmeals, the virus is excreted in saliva from day five after inoculation. Similar experiments with *C. brevitarsis* have not found ephemeral fever virus in the insects' saliva.

Life cycles

Mosquitoes breed in water trapped in plants and treeholes, in ground pools or in artificial storages. *Cx. annulirostris* lays eggs in rafts of 100 or more in ground pools. Under ideal conditions, the mosquito can develop from egg hatch to adult in 8–10 days. It feeds readily on humans, livestock, marsupials and birds. It is present at the highest densities in the late wet season or summer and the early dry season and does not breed in water temperatures below 17°C.

Distribution and dispersal

Cx. annulirostris is found over much of Australia. It is by far the most important vector of the many arboviruses infecting humans and wildlife in Australia. Its southern limits in Victoria fit those of the southern spread of ephemeral fever in major epidemics. Ephemeral fever virus has also been isolated on two occasions from *Anopheles bancroftii*, a mosquito that is found in a limited area of northern Australia.

Control

There are no practical control methods for mosquitoes biting livestock. Reducing larval breeding sites in containers, discarded tyres and shallow pools around housed animals can lower local population densities.

Chapter 5

The viruses

5.1 Bluetongue virus

Introduction

Bluetongue is a disease of sheep first delineated in South Africa and now known to be widely distributed in Africa, Asia, Australia and the Americas. Cattle, buffalo and other ruminants become infected but do not usually show symptoms. Research in South Africa showed that double or triple attacks of disease can occur, because the virus exists in many antigenic types. Initially, variants were assigned to 10 groups that did not cross-protect when used as vaccines. However, many other types of bluetongue virus have been found, and the virus is now classified into 24 serotypes numbered 1 to 24. Genetic information can be exchanged between different serotypes infecting ruminants concurrently.

Genetic analysis has been used to trace the origins of bluetongue virus serotypes. Most of the serotypes found in Australia group closely together and are quite distinguishable from those originating in Africa or the Americas. Eight bluetongue virus serotypes (1, 3, 9, 15, 16, 20, 21, 23) have been detected at various times in Australia and seem related to those in Asia. Not all the serotypes are present in any particular year. Table 5.1 shows the dates when these viruses were first isolated. Serotypes 1 and 21 seem to occur constantly in parts of Australia; each year they are found in at least one of the areas where bluetongue is active — northern Western Australia, the Northern Territory, Queensland and the northeast coast of New South Wales. In contrast, the other six serotypes appear to circulate irregularly in the Top End of the Northern Territory and possibly the far north of Western Australia. It is likely that these serotypes require regular reintroduction through infected vectors blown into Australia by the annual northwest monsoons.

Table 5.1 Bluetongue virus infection of cattle in Australia

Serotype	Year virus first isolated	Neutralising antibody in cattle	
		Northern Territory	Other States
1[b]	1979	Pre 1969[a]	Pre 1958[a]
21[b]	1979	Pre 1969	Pre 1958
20[c]	1975	1973	Nil
23[d]	1982	1982	Nil
15[d]	1982	1980	Nil
9[e]	1985	1985	Nil
16[f]	1986	1984	Nil
3[f]	1986	1986	Nil

[a] This is the earliest year that stored serum was tested for antibodies
[b] St George et al (1980)
[c] St George et al (1978)
[d] Gard et al (1985)
[e] Gard et al (1987)
[f] Gard et al (1988)

Host range

Bluetongue viruses can infect a wide range of domestic and wild animals. Antibody has been detected in several species of domestic and feral ruminants in Australia. The virus has been isolated from sentinel cattle, buffalo, sheep and goats. Antibodies to bluetongue have not been found in horses, donkeys, pigs, dogs, marsupials or humans.

Transmission and vectors

Under natural conditions, bluetongue viruses are transmitted only through insect vectors belonging to the genus *Culicoides* (Table 4.1). The most efficient vector is *C. fulvus* (62%), but it is not important in the major sheep raising areas of Australia because it is limited to the high rainfall areas of the tropics and a subcoastal habitat. *C. actoni* and *C. wadai* are found north or east of the sheep country but present no threat of spreading bluetongue virus to sheep. The other bluetongue virus vector in Australia is *C. brevitarsis*. This midge has a strong preference for feeding on cattle and breeding in cattle dung, but may be found where cattle and sheep are raised together. Its low efficiency as a bluetongue virus vector (1%) means that sheep are rarely infected. In contrast, sheep raised in areas where there are cattle can occasionally become infected with Akabane virus because *C. brevitarsis* is a very efficient vector for this virus. However, the number of sheep infected is low because little of their skin is available for feeding when compared with cattle.

Geographical distribution

The distribution of bluetongue virus in Australia, shown in Figure 5.1, is limited by season, climate and the availability of vectors. Six of the eight bluetongue virus serotypes that have been detected in Australia occur only in the far north of the Northern Territory. Serotypes 1 and 21 also cycle in this area, as well as in the north of Western Australia and Queensland, eastern Queensland and northeastern New South Wales. The surveillance achieved with sentinel herd systems will detect any expansion of bluetongue virus activity beyond the endemic areas.

Seasonal patterns

There is no evidence that bluetongue virus overwinters in cattle or sheep. In most years peak transmission in cattle in Australia occurs in late summer and autumn, as in the United States and South Africa. In the northern half of Australia most activity occurs during and shortly after the wet season. On the New South Wales coast, a distinct seasonal pattern of transmission is observed, restricted to the summer and autumn. During winter and spring, periods of 4–7 months are free from bluetongue virus transmission. In some years, there is no transmission during the summer and autumn, providing even longer virus-free periods.

Natural history

Bluetongue virus in the saliva of a *Culicoides* midge remains in the wound made by an infected midge taking a bloodmeal. The regional lymphatic system draining the skin is critical to the initial spread of bluetongue virus. Primary virus multiplication occurs in the local lymph node. The spleen is a secondary site of virus growth. Viraemia can be detected in cattle at three days after infection; in natural infection in Australia it usually persists for less than eight weeks, and often for less than four weeks.

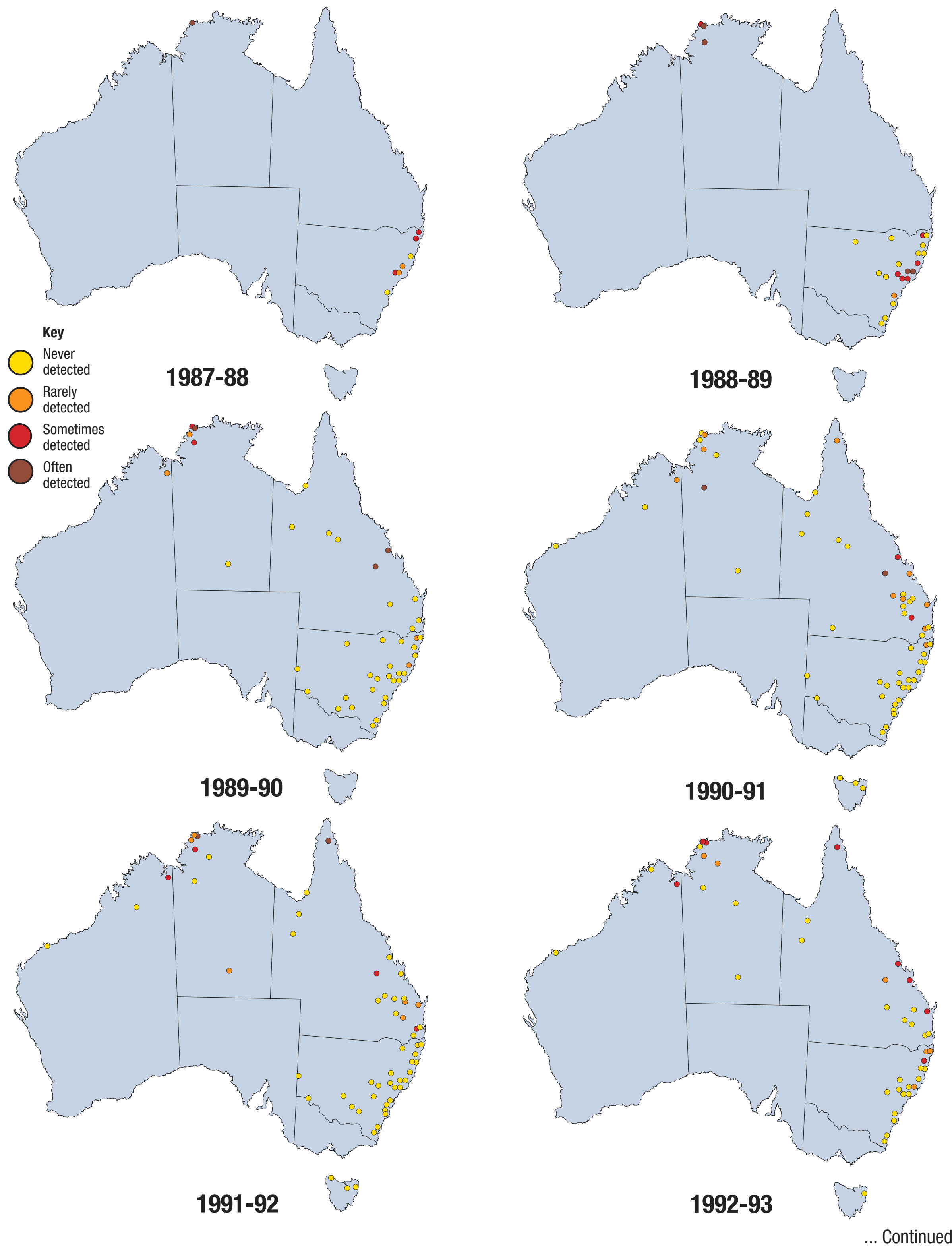


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Figure 5.1 Distribution of bluetongue virus seroconversions from 1987–88 to 1998–99, as detected by NAMP sentinel herds. Spots represent sentinel herd sites; colours indicate the number of seroconversions recorded at that site in each year (see key).
Source: NAMP national database.

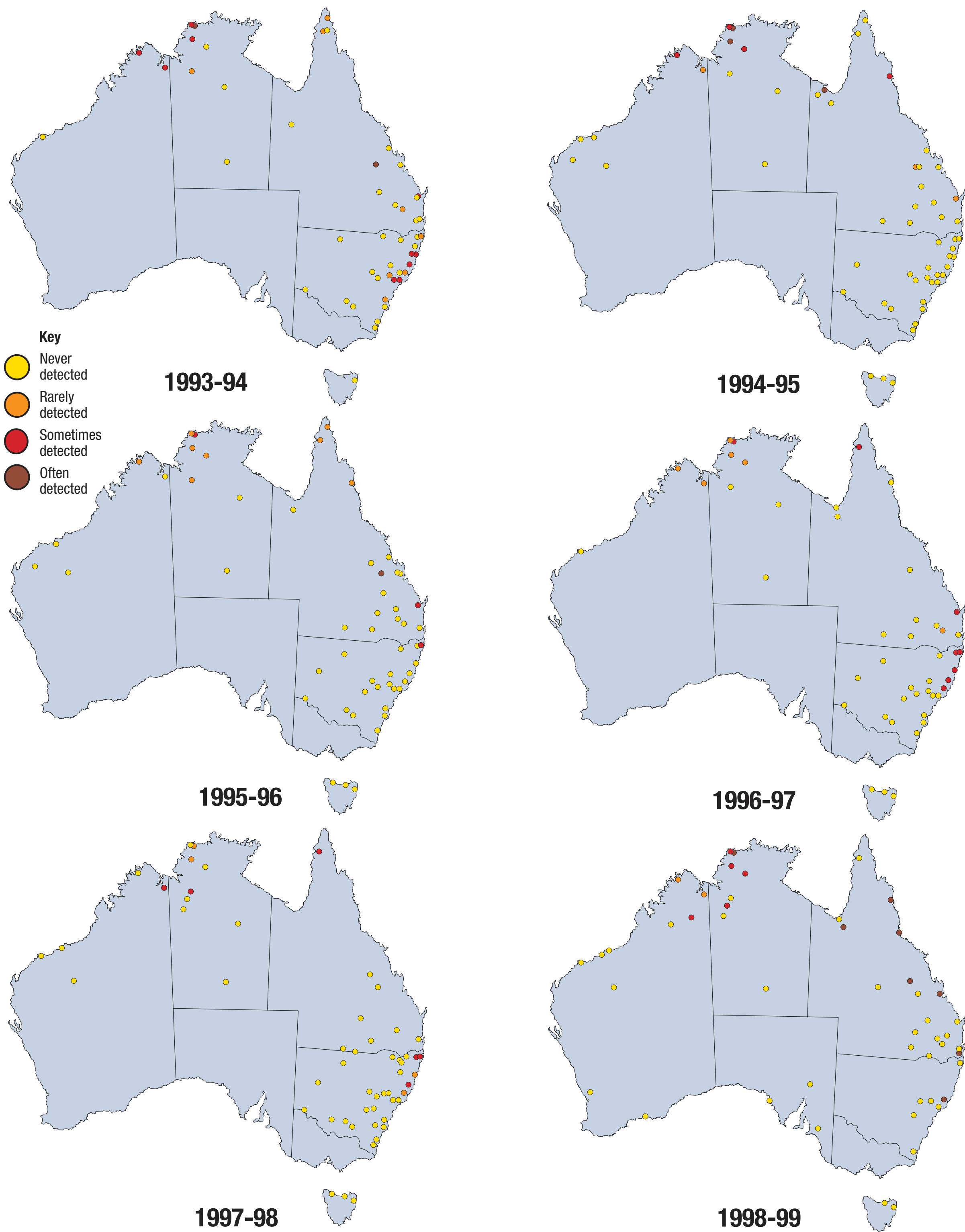

Figure 5.1 (Continued)

In the initial stages of infection, virus is associated with all blood cell types except granulocytes. The persistence of bluetongue virus is directly related to the life of the particular cell type in the circulation. Virus particles attach passively to the wall of red blood cells, but cannot multiply in these cells, which lack a nucleus. Neutralising antibodies appear at 6–8 days after infection and may persist anywhere from six months to the lifespan of the animal. The coexistence of virus and specific antibodies in the bloodstream is not unusual in the acute stages of infection. Early in infection, the virus and homologous antibodies coexist in the bloodstream, facilitated by the close association of bluetongue virus with the envelope of red blood cells, which possibly causes steric hindrance of immune clearance mechanisms. Virus genetic material can be detected by polymerase chain reaction (PCR) weeks after infectivity disappears, for as long as subviral material remains in the vascular system. However, there is no evidence that insect vectors can be infected with virus from cattle positive by PCR for viral nucleic acid but negative for live virus. Infectious virus is only found for a maximum of about 60 days, and then only in a very small proportion of infected animals.

Bluetongue virus may be found in the semen of older bulls during early viraemia, induced experimentally with cell-culture-adapted virus, but rarely after natural infection. There appears to be little risk of transmission by infected semen following natural infection, and washed embryos are safe. Body exudates and infected meat do not spread bluetongue virus.

The concept that cattle can be infected for life has been largely discredited. The precise time at which cattle cease to circulate enough virus to infect *Culicoides* midges is uncertain, but it is probably much less than the eight weeks that live virus can be cultured from blood. Sheep have a shorter period of viraemia, perhaps due to the shorter survival time of red blood cells, especially in young sheep.

The experimental use of virus strains that have been altered by passage through embryonated eggs, tissue cultures or the brains of suckling mice has generated some confusion over the biology of bluetongue viruses. For example, wildtype, unadapted virus is not usually excreted in the semen of bulls and does not cause congenital defects in lambs.

Pathogenesis and pathology

The clinical effects of bluetongue in sheep can range across a broad spectrum, from subclinical illness to sudden death. The disease is characterised by several stages, with an incubation period of 5–6 days followed by an abrupt rise in body temperature, cessation of appetite and general depression. The virus affects blood vessels, resulting in inflammation and haemorrhages above the hoof, swelling of the face, ears and legs, fluid in the lungs and, in extreme cases, congestion of blood in the tongue (blue tongue).

Clinical importance

There is no evidence that bluetongue causes clinical illness in cattle in Australia. Bluetongue is an important clinical disease of sheep in some countries with a temperate climate due to distance from the equator or altitude. In the tropics, bluetongue is uncommonly seen in indigenous sheep. However, major losses may occur if susceptible sheep are imported from areas that are free from the bluetongue virus into infected countries or zones. Southern Africa, the Middle East, the United States of America, China and India are the principal areas where bluetongue virus causes economic loss from disease. There is no evidence of any clinical disease associated with bluetongue virus infection in livestock in Australia.

Diagnostic methods

Bluetongue infection of animals needs to be confirmed by laboratory tests. If necessary, live virus or viral fragments can now be detected in a blood sample by PCR in about one day. Identification by isolation of virus in embryonated eggs and tissue culture takes from two to four weeks.

Neutralising antibodies can be detected from about one week after infection and may remain detectable for anywhere from six months to the lifespan of the infected animal. A specific and rapid bluetongue blocking or competition enzyme-linked immunosorbent assay (ELISA) has been developed to avoid the false positives caused by older, more broadly reactive tests. The ELISA can be completed in a few hours.

Control

Control of bluetongue disease in sheep is more complex than control of Akabane and ephemeral fever because there are multiple serotypes of virus and many different animal hosts. In Australia, bluetongue viruses circulate mostly in cattle because the principal *Culicoides* vectors breed only in cattle dung.

Large-scale control of *Culicoides* species implicated as vectors of bluetongue disease is currently not feasible. *C. brevitarsis* is the only vector of bluetongue viruses with a distribution that overlaps sheep-rearing areas in Australia. Infection of sheep in Australia is uncommon and has not been associated with disease. If vectors need to be controlled, the population of *C. brevitarsis* can be reduced locally by treating cattle with systemic ivermectin. The drug renders the dung toxic for *Culicoides* larvae for 3–5 weeks. Placing cattle under cover reduces attack by *Culicoides* but its usefulness is limited.

Vaccination has been used in some countries to protect sheep from clinical bluetongue. As disease has not occurred in Australia, bluetongue vaccines are not available.

5.2 Akabane virus

Introduction

Akabane virus is a member of the Simbu group of viruses found throughout Africa and Asia. The discovery that Akabane virus caused foetal defects in cattle and sheep led to increased research into arboviruses. Akabane virus was isolated from insects in Japan and Australia some years before it was associated with disease. Evidence for the association came from Japan, where Akabane virus was isolated from an aborted foetus. Also, antibodies to the virus were found in a high proportion of cattle that had borne deformed calves and in newborn deformed calves bled before suckling. Commonwealth Scientific and Industrial Research Organisation (CSIRO) scientists learnt of these findings from a visiting Japanese scientist just before a major Akabane epidemic in New South Wales in 1974. The association between the virus and the disease was rapidly confirmed by New South Wales veterinarians and CSIRO scientists.

Antigenic variation of Akabane virus occurs in nature but the differences are relatively minor and are not sufficient to define specific serotypes. Strains vary in their capacity to produce deformities in animals under experimental conditions.

Host range

In Australia, Akabane antibodies have been found in cattle, buffalo, sheep, camels, deer, goats, horses and dogs but not in pigs, marsupials or humans. Akabane virus has been isolated from sentinel cattle and sheep. Foetal deformities have been found only in calves, goat kids and lambs in Australia.

Transmission and vectors

Akabane virus is transmitted between domestic animals by insect vectors. The only certain vector is *C. brevitarsis*. Akabane virus has been isolated from *C. wadai* but no further studies have been carried out to confirm it as a vector.

Geographical distribution

Akabane virus probably entered the Northern Territory from Indonesia, the virus not having been found in Papua New Guinea before 1992. Possibly, *Culicoides* carrying the virus were blown across the Timor Sea in sufficient numbers to establish an ecological link to cattle and buffalo. The distribution of the virus, shown in Figure 5.2, varies from year to year in a complex fashion; on very rare occasions it extends almost to the limits of the distribution of *C. brevitarsis*.

Seasonal patterns

The virus may be spread in northern tropical areas during the dry season from early spring. In the more southern parts of Queensland and New South Wales it has a distinct seasonal pattern of spread, with transmission usually restricted to summer and autumn and infection ceasing when the first cold weather occurs.

Natural history

The key to unravelling the natural history of Akabane virus was its isolation from insects rather than from animals showing the effects of virus infection. Once the virus became available, specific tests were developed and used to establish the time of year when infection could be expected. In a major experiment at Camden, New South Wales, sheep were mated so that they would be in mid-pregnancy when natural infection was expected. Once Akabane virus was active in the flock, the pregnant sheep were killed and the foetuses examined for physical defects and the presence of virus. This approach, together with experimental infection of pregnant sheep and cattle at various stages of pregnancy, was used to determine the characteristic effects of the virus in newborn calves and lambs.

When an infected female *Culicoides* bites a pregnant cow or ewe, Akabane virus enters the wound in the saliva. There is probably some local multiplication of the virus as it moves through the lymph to the bloodstream, although this has not been directly confirmed. Once in the blood, the virus is distributed throughout the body and multiplication continues. The maternal placenta is very rich in blood vessels and the cells of its various layers support replication of Akabane virus. The virus crosses the foetal layers of the placenta and enters the foetal circulation, causing abnormalities related to the stage of gestation at which infection occurs.

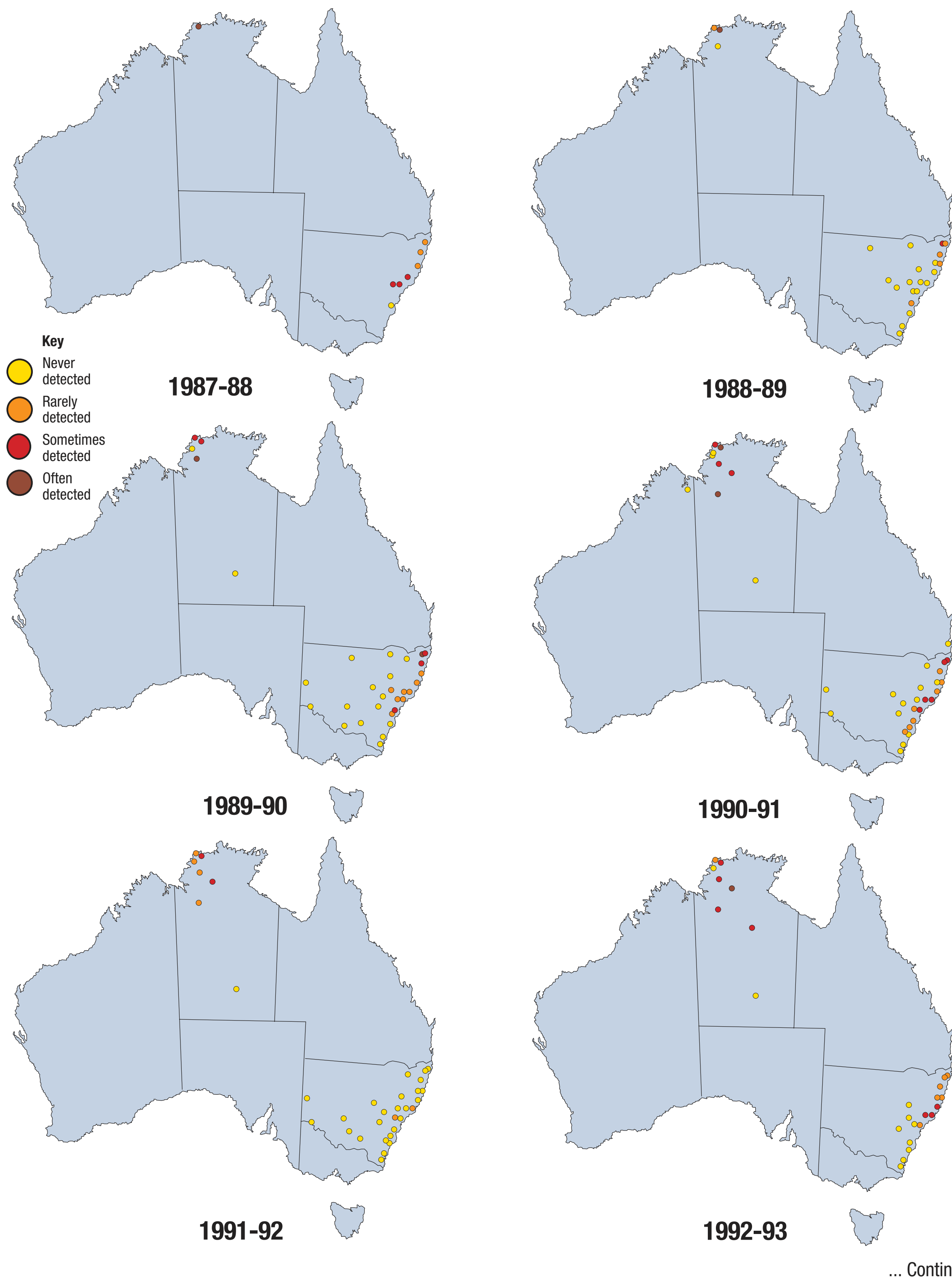

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Figure 5.2 Distribution of Akabane virus seroconversions from 1987–88 to 1998–99, as detected by NAMP sentinel herds. Spots represent sentinel herd sites; colours indicate the number of seroconversions recorded at that site in each year (see key).
Source: NAMP national database.

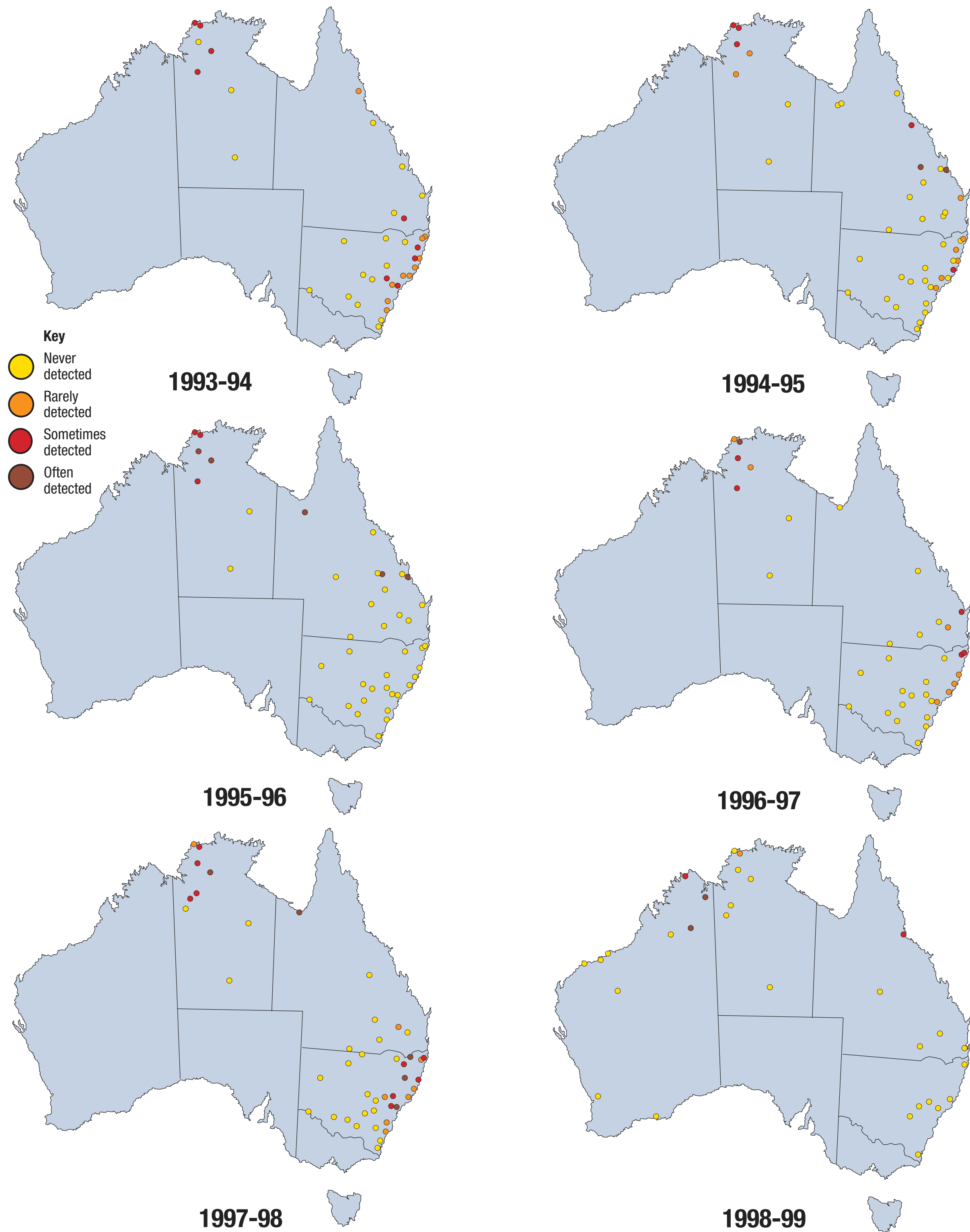

Figure 5.2 (Continued)

In the cow, Akabane virus is normally detectable in the blood for 3–4 days after infection. Neutralising antibodies are detectable 1–2 days after viraemia, and clear the virus from the circulation. In pregnant cows, the placenta and foetus may become infected. Circulating antibodies do not normally pass to the foetus, so the virus multiplies in rapidly dividing foetal cells and causes damage until the foetus produces its own antibodies. The virus in the foetus is inaccessible to insects and is not transmitted from this source.

Pathogenesis and pathology

The pathology of the effects of Akabane virus had been well studied before the causative agent was identified. Epidemics in New South Wales in 1956, 1974 and 1983 have been recorded in detail. In each outbreak a similar range of pathology occurred.

In a survey of natural infections in New South Wales, 17% of calves born to cows infected between 76 and 224 days of gestation were defective. Defects are regularly encountered in 25–40% of calves in a seasonal calving herd where infection occurs at critical stages of pregnancy. The proportion of foetuses infected is strain related and varies under experimental conditions from 42% to 83%, depending on the strain.

The virus has the greatest effect on tissues that are undergoing most rapid development and differentiation at the time of infection. The nervous system is most affected in the calf. Damage can vary from microscopic, through cavitation of the brain, to complete absence of the cerebrum (hydranencephaly). When the effects are very severe, only the brain stem and the membranes covering the brain remain intact. As the brain stem controls basic functions such as circulation, breathing and limb movement, most affected calves are born alive. Limb and spinal defects are also characteristic of Akabane virus infection. One or more limbs may be fixed in flexion or extension (arthrogryposis) and the spine may be bowed or twisted. When calves are affected with severe arthrogryposis, obstetric complications are common. If early assistance is not provided, cows may suffer complications and even death. The full spectrum of abnormalities may be seen in herds with year-round or extended calving periods, whereas those with a short mating period may have only one type of abnormality. If the foetus dies, abortion can follow, usually at about 5–7 months of gestation.

The effects of Akabane virus infection and the relative frequency of particular defects at particular stages of gestation are summarised in Table 5.2. The time boundaries shown are not rigid but provide a guide to the effect of infection at particular stages of pregnancy.

Table 5.2 Relationship between stage of gestation and effect of Akabane virus infection in calves

Stage of pregnancy at infection	Effect of infection with Akabane virus
Conception–77 days	No damage known
78–103 days	Hydranencephaly
104–172 days	Arthrogryposis
173–224 days	Polioencephalomyelitis
225 days–birth	Encephalitis

In sheep and goats, the foetus is most susceptible from 28 to 56 days. Due to the short gestation, affected lambs or kids often show several types of defects.

Certain species of animal become immunocompetent (able to mount an immune response, including antibody production) before birth. Calves produce antibodies from approximately 90 days of gestation and lambs from 65 to 70 days. Once antibodies appear, Akabane virus is rapidly cleared from the foetus; thus the virus is not found in calves or lambs with foetal defects born near or at full term. However, the damaged neural tissues are never repaired and the defects are evident at birth.

Clinical importance

Akabane virus appears to be harmless to nonpregnant animals. Due to the time interval between infection of sheep or cattle in early pregnancy and the birth of their offspring, infection by Akabane virus occurs in one season but its effects are observed in another. For example, in New South Wales the peak of infection is in the autumn and the peak of abortions or birth of deformed calves occurs in the spring. The deformed newborn calves are free of virus and do not perpetuate infection.

In endemic areas, losses may be sporadic because not all heifers are infected before becoming pregnant. However, the disease caused by Akabane virus is clearly observed in epidemic years, when it spreads beyond the fringes of its normal distribution in Queensland and New South Wales and affects many thousands of calves. Disease is uncommon in sheep because there are few sheep in the main vector areas and few sheep are pregnant when vectors are active.

Diagnostic methods

The causative virus cannot be isolated from calves or lambs that are obviously suffering tissue damage unless they are aborted well before term. Pathological changes are a more useful diagnostic indicator. The simplest and most direct method is to open the skull and check the brain visually for lesions. Major damage to the cerebrum can be observed directly but the cerebellum is not affected. Brain tissue and spinal cord can be fixed in 10% formalin solution for histopathological examination. Spinal abnormalities may be localised in calves with arthrogryposis and limited to one limb or a few joints.

Whole blood taken from a calf or lamb that has not suckled colostrum is the most appropriate specimen to establish Akabane as the causative agent. If the animal is killed after collection of the blood, its abomasum can be checked for absence of curdled milk. Pericardial fluid can be used to test for antibodies to the virus if animals are dead at birth. Antibodies to Akabane virus can be detected by virus neutralisation or by ELISA. As other viruses in Australia can invade and damage ruminant foetuses, laboratory tests are needed to determine which virus is involved.

Control

It is not feasible to control transmission of the virus by killing the vector, *C. brevitarsis*. A highly effective killed vaccine was available but is currently unavailable, due to commercial considerations by the manufacturer.

5.3 Bovine ephemeral fever virus

Introduction

Bovine ephemeral fever (BEF) came to notice in the latter part of the 19th century in southern Africa and Egypt. The disease was also known in Asia, in draft cattle and water buffalo, long before it was described in Australia in 1936 as a new disease causing a spectacular epidemic. Almost 100 years ago, in southern Africa, infection was successfully transmitted from one cow to another by blood transfer; it was suspected that it was transmitted by a flying insect. Cattle in good condition and producing high milk yields suffer the worst effects of ephemeral fever; rest is vital to full recovery, especially in draft animals. As early as the 19th century, there were warnings against giving medicaments by mouth to acutely ill cattle.

In Canberra in 1939, Ian Mackerras and his wife, Josephine, completed a classic study of the disease. Their thorough investigation delineated the principal parameters of the disease. The study was especially notable because cattle were subinoculated to define the length of viraemia, identify the blood cells carrying the virus and compare routes of inoculation. Ice was used instead of refrigerators and the most sophisticated laboratory tools available were small, low-speed centrifuges and monocular microscopes. The Mackerras' findings have been extended but not faulted.

The virus has gone by many names, but the term bovine ephemeral fever (BEF) virus is now recognised internationally. Comparisons between Australian, African and Chinese strains have shown cross-protection, suggesting homogeneity. However, research in Australia, using virus sequencing and monoclonal antibodies, has revealed wide genetic variation. These studies have not been extended internationally.

South African researchers were the first to isolate BEF virus from the blood of cattle, by passage through the brains of suckling mice in 1966. The virus appeared to lose antigenicity as well as virulence for cattle during passage and was thus a poor candidate for a vaccine. The normal route to administer a vaccine is under the skin or into a muscle. Even highly virulent BEF virus, in freshly drawn whole blood, rarely produces disease when administered by skin or muscle rather than a vein. These findings have only recently been explained and are discussed under 'Pathogenesis and pathology'.

Host range

Disease described as ephemeral fever has been reported only in cattle and water buffalo, although many ruminants have antibodies to the virus. Cattle are the only livestock in which the disease has been observed in Australia. In Asia, introduced water buffalo suffer the disease, but no clinical cases have been noted in water buffalo in Australia; although natural infection occurs no symptoms are seen. Serological surveys in Africa have found antibodies in a range of species of deer and antelope. Some of these data may be unreliable because other viruses share antigens with ephemeral fever virus. In Australia, neutralising antibodies found in introduced deer have been confirmed as specific for ephemeral fever, but domesticated deer in Queensland have not shown signs of the disease when it has occurred in cattle pastured nearby.

Transmission and vectors

Ephemeral fever virus is naturally transmitted only by insect vectors. Numerous experiments with susceptible cattle housed with sick animals in insect-proof buildings have failed to demonstrate any transmission by contact. Meat of infected animals poses no risk, for two reasons. First, the virus is susceptible to the low pH produced by lactic acid formation in muscle postmortem; second, the virus cannot be transmitted by mouth. Many mosquitoes and biting midges have been considered as vectors. The first isolation of ephemeral fever virus, from a mixed pool of *Culicoides* species in 1974, focused attention on *Culicoides*. However, as discussed in 'Pathogenesis and pathology', mosquitoes are more likely vectors. Ephemeral fever virus has been isolated from a mixed pool of culicine species of mosquitoes and twice from *Anopheles bancroftii*, which has a restricted distribution in northern Australia. The most important vector is probably *Cx. annulirostris*, but *A. annulipes* may also be involved. Improved virus detection methods are now available that could settle this point.

Geographical distribution

Ephemeral fever occurs in much of Africa, Asia and Australia but not in Europe, the Americas, Papua New Guinea or the Pacific Islands. It is not known how or when the virus entered Australia but it is presumed to have done so from Indonesia. The disease first came to notice in 1936, with a major epidemic that moved from the Northern Territory to New South Wales and Western Australia over two successive summers. Major epidemics followed a north-to-south pattern in 1955–56, 1967–68, 1970–71, 1972–74 and 1975–76. Subsequently, there was a more regular spread each summer, with the virus appearing to overwinter in southern areas. Figure 5.3 shows the distribution of the virus from 1987–88 to 1998–99. In general, the virus spreads from infected to adjacent, uninfected areas. On rare occasions, cattle introduced from infected areas by road transport may have caused limited outbreaks. The southernmost limit, reached in 1968, has not been observed since.

Seasonal patterns

Ephemeral fever can occur in the spring, summer or autumn in northern Australia. Where there is a cold winter, such as in New South Wales and parts of southern Queensland, the disease is usually observed only in summer and autumn. An experiment was conducted from 1974 to 1977 along the course of the Flinders River in Queensland to determine whether ephemeral fever virus circulated in cattle at a low level in the dry season. The virus passed through the observational groups in three successive summers but not in the winter, although susceptible cattle were available to vectors. Occasional, out-of-season local epidemics occur in north Queensland when the normal winter dry season is broken by abnormal rain. In the past, the behaviour of ephemeral fever could only be predicted locally, during an outbreak. Now, regularly monitored sentinel herds allow predictions to be made regionally and in advance of an outbreak.

Natural history

Ephemeral fever virus enters the bloodstream directly with mosquito saliva. The early stages of multiplication are still uncertain. The virus can be found intermittently in the bloodstream during the incubation period but is always detectable before the first febrile period. Thus, it can infect a new wave of insect vectors within 2–4 days of transmission.

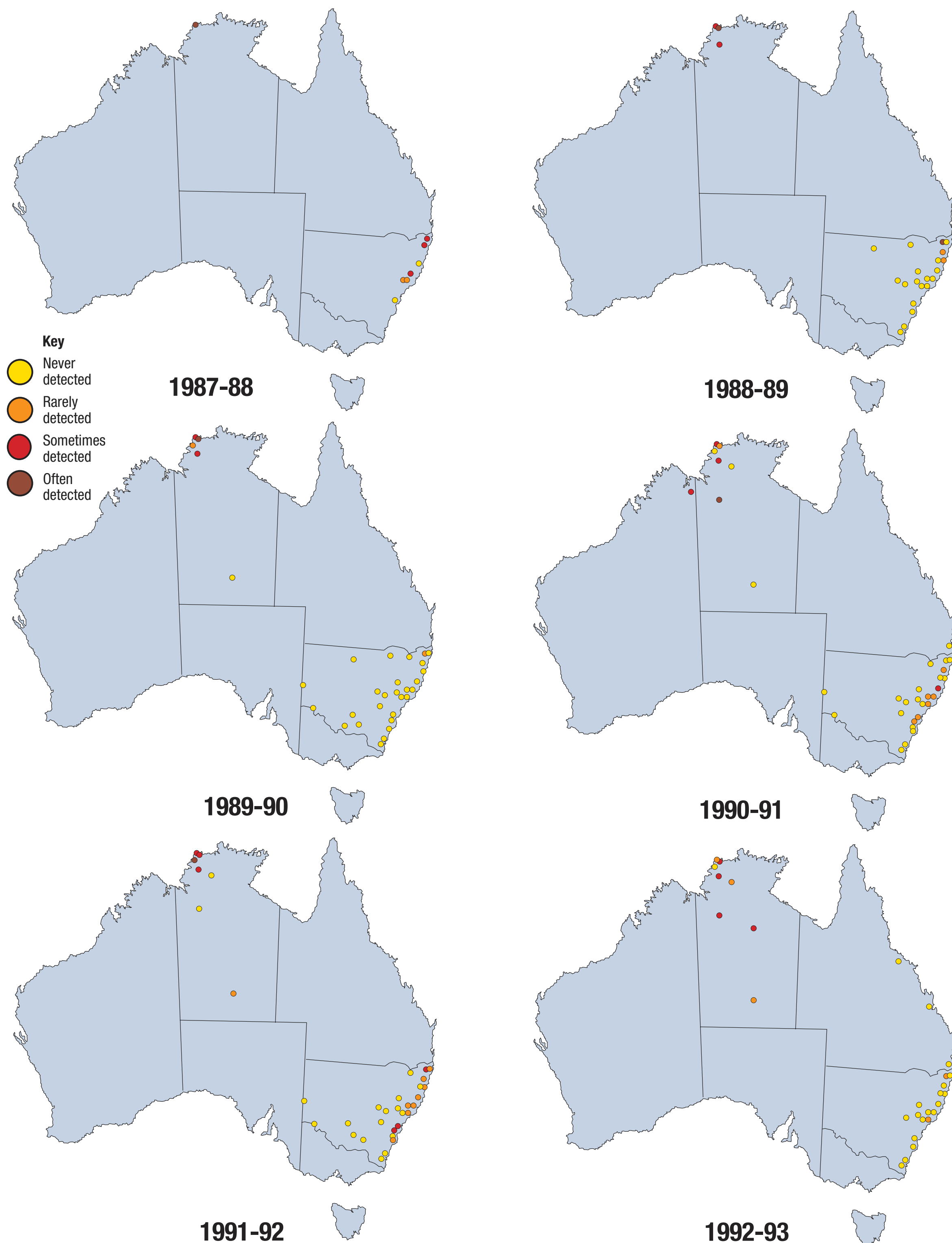

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Figure 5.3 Distribution of ephemeral fever virus seroconversions from 1987–88 to 1998–99, as detected by NAMP sentinel herds. Spots represent sentinel herd sites; colours indicate the number of seroconversions recorded at that site in each year (see key).
Source: NAMP national database.

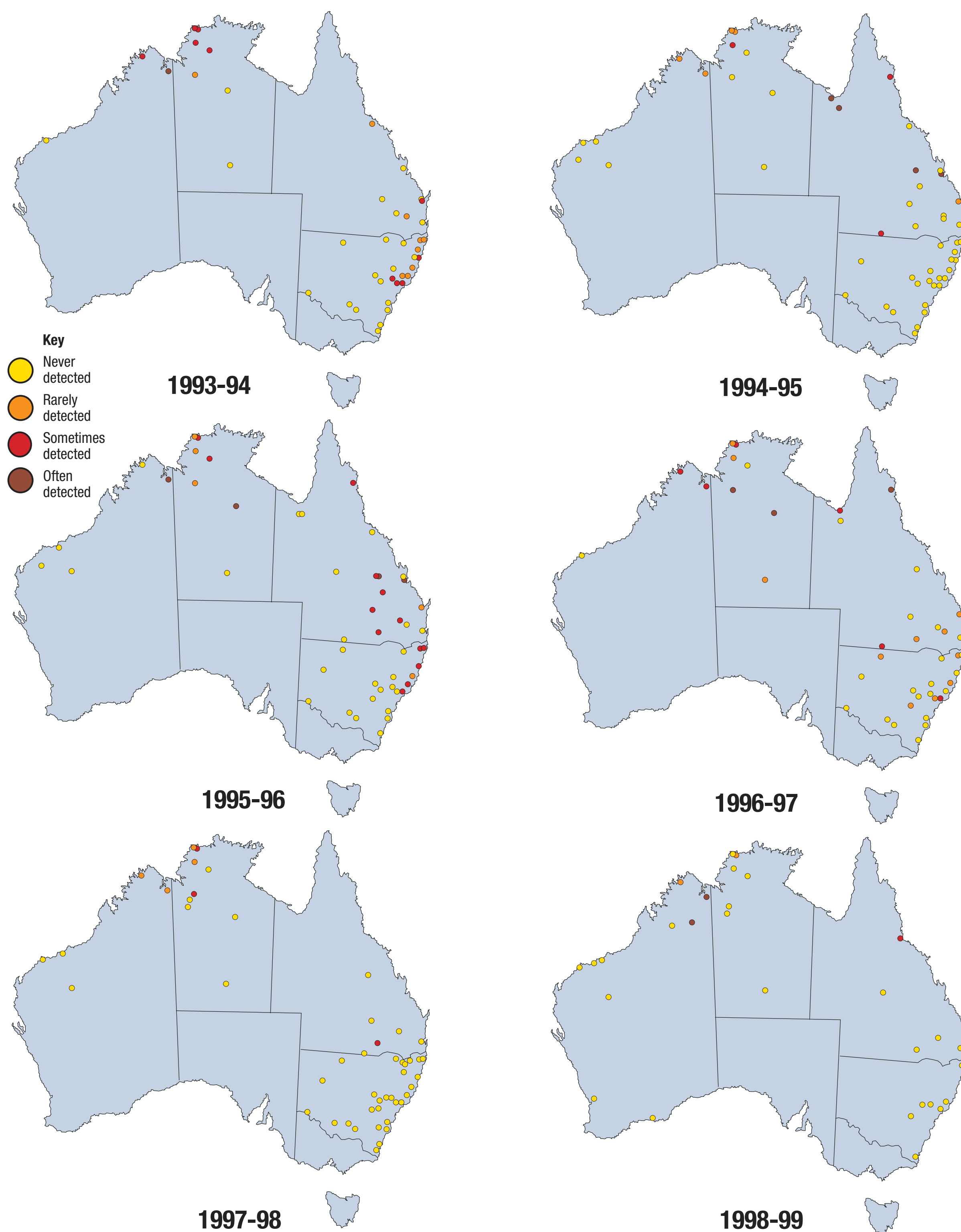

Figure 5.3 (Continued)

Pathogenesis and pathology

The pathogenesis of ephemeral fever has been studied for many years, especially in South Africa and Australia. A problem in studying the disease is that its clinical course is unlike that of any other insect-borne disease. Ephemeral fever virus has to be delivered directly into the bloodstream to transmit the disease experimentally. In nature, this occurs when a mosquito inserts its mouthparts into a venule. The virus multiplies within the bloodstream, and is carried into the tissues in white blood cells. Virus is detectable before the first febrile response, which occurs 3–7 days after virus is injected. The first episode of fever lasts for approximately one day. During this period, infected cattle appear only slightly depressed, but the milk yield of lactating cows drops abruptly. A second temperature rise may occur at the end of the first episode or after a delay of up to one day. It is in the second or later phases that the characteristic symptoms are seen in varying degrees of severity.

Ephemeral fever virus circulates during fever, closely associated with neutrophils. The life of neutrophils is measured in hours, not months as for red blood cells. Inflammation of the walls of small blood vessels allows fibrin-rich fluid to move into the abdomen, chest and joint spaces, carrying virus and neutrophils with it. The virus-containing neutrophils do not return to the bloodstream.

Cattle with ephemeral fever show symptoms varying from mild to fatal. Symptoms tend to be less severe in young cattle than in mature animals, and may be as mild as loss of appetite, unwillingness to move, slight lameness and loss of milk production. In more severe cases, there may be complete loss of ruminal function, disinclination to drink, severe depression, ocular and nasal discharges, subcutaneous oedema and recumbency. Paralysis affecting the hindquarter or all limbs may follow. In the most severe cases, all reflexes are lost and breathing is extremely rapid. Signs of pneumonia, air under the skin and complete paralysis may precede death.

The symptoms in most cattle end abruptly on the second or third day of illness. Recovery occurs 2–4 days before antibodies are detectable. Once specific antibodies rise to a sufficient level, there is no recurrence of ephemeral fever virus in the bloodstream. Cattle with even low levels of specific antibody resulting from natural infection are completely resistant to experimental infection.

Spontaneous and rapid recovery can occur at almost any time, even with apparently severe paralysis. Most of the symptoms are due to inflammation associated with the infection rather than to direct action of the virus. Thus, anti-inflammatory drugs can completely prevent symptoms and temperature rise if given very early in infection and at intervals throughout the expected course of the disease. Anti-inflammatory agents can also cause rapid improvement if given once fever is established. Untreated cows in lactation do not return to full milk and may stop lactating. Abortion occurs in about 2% of pregnancies and is usually confined to cows in the later stages of gestation. In beef cattle, weight lost is regained in 1–2 months.

Mortality (average 1.5%) is more likely in high-producing dairy cattle or beef cattle in fat condition and under very hot conditions. As many affected cattle cannot drink or are temporarily too disabled to reach water, dehydration contributes to fatalities.

Comparatively few cattle have been examined after dying naturally from ephemeral fever. However, the changes seen postmortem are consistent with a generalised inflammation. There is usually straw-coloured fluid in the joint capsules, chest and abdominal cavities. Pneumonia is a common complication. Haemorrhages on the heart and other organs are uncommon. Microscopic haemorrhagic areas can be found in the lungs.

Clinical importance

Ephemeral fever virus causes regionally significant ephemeral disease during epidemics but is of little overall national economic importance. The losses are due to mortality in dairy cattle and marketable beef cattle; loss of milk production; loss of weight gain; disruption to dairy routines or husbandry activities in beef cattle; and treatment, vaccine and veterinary costs. Untreated bulls can become infertile for several months.

Diagnostic methods

Ephemeral fever is usually diagnosed from the clinical history of a herd when cattle are in various stages of disease, from early fever to recovery. The simplest confirmatory technique is to take a blood sample, make a smear and allow the balance of the sample to clot. The fixed smear is stained and used to determine the neutrophil percentage. If the neutrophil count does not exceed that of the lymphocytes and there are no band forms, then the disease is not ephemeral fever. However, a high neutrophil count does not prove infection with ephemeral fever. Although BEF virus is virtually the only virus causing a neutrophil response in cattle, several bacterial diseases also increase neutrophil numbers. If the animal is infected with ephemeral fever virus, the blood may not contract normally during clotting and the clot will be streaked with white fibres. This clotting phenomenon also occurs in severe milk fever.

If confirmation of diagnosis is required, the preferred technique is to collect paired sera, with a convalescent sample taken 14–21 days after the onset of disease. The paired samples should be tested concurrently by virus neutralisation or ELISA to demonstrate seroconversion or significantly rising antibody levels. Alternatively, virus isolation may be attempted, but this is less successful. A blood sample is collected during the very early stages of fever with lithium–heparin anticoagulant. The sample is centrifuged and the white cell layer inoculated into flasks containing a monolayer of mosquito cells derived from *Aedes albopictus*. Cells are incubated at 36°C for 13 days, then checked with specific fluorescence tests for the presence of ephemeral fever virus. Results can now be obtained rapidly using more advanced techniques, such as PCR, for virus detection.

Control

It is not feasible to interrupt insect transmission with currently available technology, so control measures must be applied to cattle. Vaccination with an attenuated virus produces lifelong immunity in most virus diseases. However, live-virus vaccines have produced only short-term immunity to ephemeral fever, even when adjuvants are added. The problem appears to be that multiplication of virus is limited and occurs only around the vaccination site. Two doses spaced at 2–4 weeks are necessary to initiate immunity, and boosters are essential. The live vaccine has the disadvantage that a cold chain must be maintained from factory to administration. Also, the vaccine is in two parts and must be reconstituted just before use.

There is now an inactivated Australian vaccine with the same dose of virus, inactivated by formalin. It does not require premixing and is administered in two doses spaced at 3–4 weeks apart. The regime recommended for the boosters to maintain immunity depends on the level of disease threat. An experimental subunit vaccine developed from the surface protein of ephemeral fever virus has produced a sterile immunity to challenge with virulent virus but has not been manufactured on a commercial scale.

Appendix 1
KnowledgeBase

Information generated by Australia's National Arbovirus Monitoring Program (NAMP) is gathered, managed and reported through a national information system. The NAMP database is divided into two sections. The first holds quantitative data gathered through vector trapping and sentinel herds. The second part contains descriptive data based on the observations, experience and understanding of the state coordinators. This descriptive data provides a backdrop to the quantitative data, and captures factors that cannot be recorded in a traditional structured database. The descriptive database is known as the KnowledgeBase. This appendix contains an extract of the KnowledgeBase from the late 1980s through to 1999.

It needs to be emphasised that bluetongue disease is not seen in Australia, despite the presence of bluetongue viruses in parts of the north and east of the continent. Bluetongue viruses are not found in southern Australia or large, well-defined parts of central and northern Australia. Where 'incidence' is referred to in the section of the KnowledgeBase referring to bluetongue, it means the incidence of seroconversions in cattle.

Akabane virus

New South Wales

Normal features of Akabane virus transmission in NSW

Akabane virus transmission in New South Wales (NSW) is usually confined to the coastal regions of the state, from the Queensland border in the north, extending just south beyond the Hunter Valley. The virus will usually be spread inland to the eastern fall of the Great Dividing Range, with the western limit being defined by altitude as cooler temperatures influence the spread of *Culicoides* vectors. Spread beyond these limits, to either the west or south, occurs only under very favourable conditions, particularly mild temperatures and, to a lesser extent, relatively high rainfall. Conversely, very low rainfall can reduce transmission and distribution within the 'endemic' region. Virus transmission occurs only during the warmer summer and autumn months. In the far northern districts, infections usually occur in late January–February; with very favourable conditions, they may occur in December. On the lower North Coast and in the Hunter–Manning regions, transmission commences in February–March. Transmission may continue in the endemic region until late May–early June but is not observed during the colder months of winter and spring. If the virus extends beyond the endemic area under extremely favourable conditions for the vector, seroconversions may be observed in inland and southern areas in March–April. Within the Akabane virus endemic coastal region, transmission is usually observed each year. The

incidence of infection within the endemic coastal region is usually very high at all locations, with most (> 80%, commonly 100%) sentinel animals seroconverting. At the far extremities of the region, or in less favourable years, these seroconversion rates may be reduced, with 50–70% of animals becoming infected. Due to the very high incidence of spread annually, female cattle become immune before they reach breeding age and disease is rarely observed in the endemic region. Disease is observed in NSW only when either non-immune animals are introduced to these endemic districts or the virus is spread beyond the usual limits. Sporadic cases of disease are observed at intervals of 2–3 years. Major epidemics of disease occur at intervals of 8–10 years. Comments relevant to transmission in specific years are as follows.

Akabane in NSW

Year	Description
1932	First documented epidemic (Hindmarsh 1937).
1951, 1954–55	Cases on the South Coast and in the Central West (Blood 1956; Christie 1956).
1960, 1964, 1968	Outbreaks on the South Coast (Bonner et al 1961).
1974	Virus transmission during the late summer–autumn of 1974 extended far beyond what are now recognised as the usual southern limits for the vector. Although the spread was not actively monitored (there was no known association between Akabane virus and disease in calves until about August 1974), its spread was eventually documented after the occurrence of the largest outbreak of Akabane virus-induced congenital defects in calves to be recorded in Australia (more than 8000 cases). Disease was recorded along the entire South Coast and Southern Tablelands of NSW and extended into northern coastal Victoria (Gippsland). (Della-Porta et al 1976; Hartley et al 1977; Shepherd et al 1978)
1976–78	Outbreaks of disease reported on the New England Tablelands of NSW (Coverdale et al 1978; Everett 1979).
1979–81	***Transmission***. Monitoring was confined to the Hunter Valley. The Akabane transmission pattern was relatively normal, with infection at each of the four monitoring locations through the valley. Seroconversions were observed between February and April from Tomago on the coast to Scone in the upper Hunter Valley. ***Incidence***. The incidence of Akabane infection was lower than usual in 1980 and 1981, with 70–90% of sentinels seroconverting at Tomago and Paterson but only 20–70% at Singleton and Scone. ***Disease.*** Not recorded. ***Features***. A relatively normal Akabane pattern except for the lower incidence, almost certainly due to the severe drought conditions from late 1980 onwards.
1982	***Transmission***. Sentinel monitoring was confined to the Hunter Valley but followed by a cross-sectional survey. There was an unusual pattern of Akabane transmission, with infection restricted to the lower Hunter Valley. Seroconversions were not observed beyond Paterson, where transmission was delayed until April. The cross-sectional survey indicated that the virus did not spread westwards beyond Branxton (Kirkland et al 1983). ***Incidence***. In areas where infection occurred, the incidence of Akabane infection was normal. ***Disease***. Not recorded. ***Features***. An unusual Akabane pattern, with significantly reduced spread.

Akabane in NSW (cont'd)

1983	***Transmission***. Sentinel monitoring was confined to the Hunter Valley and Jamberoo on the South Coast but was followed by a cross-sectional survey over a large part of the state. The Akabane transmission pattern was quite unusual, with very rapid spread through the Hunter Valley. The subsequent cross-sectional survey and disease outbreak showed extension westwards as far as Trangie and south to Cowra, Crookwell and Goulburn. There was also spread along the Northern Tablelands and northwest slopes. The onset of seroconversions in the Hunter Valley was relatively normal, with infection commencing in late February, but spread was very rapid, moving from Paterson westwards to Scone in less than two weeks. Seroconversions occurred at Jamberoo in early April. ***Incidence***. The incidence of Akabane infection was high (100%) at all locations where infection is usually recorded. In the cross-sectional survey, the prevalence was high in most locations and gradually declined from about 80% in the Central Tablelands to 7% in the midwest of the state at Trangie. ***Disease***. A severe outbreak was observed in the mid-upper Hunter Valley and throughout the Central West, Northern Tablelands and slopes and upper South Coast–Illawarra region. ***Features***. There was a spectacular, rapid spread of virus, leading to a disease outbreak over a large part of the state following cessation of the drought conditions in late 1982–January 1983.
1984	***Transmission***. Sentinel monitoring was confined to the Hunter Valley. The Akabane transmission pattern was unusually late, with gradual spread through the Hunter Valley in May and June. Akabane transmission appeared to have been delayed due to very early (February at Paterson) spread of Douglas virus. ***Incidence***. The incidence of Akabane infection was moderate to low (70% at Paterson and 30% at Scone) at all locations where infection is usually recorded. ***Disease***. No disease was observed. ***Features***. Very late transmission of Akabane virus with a lower incidence than usual.
1986	***Transmission***. Sentinel monitoring was confined to the Hunter Valley (Paterson and Singleton) and Camden. A most unusual season, with no Akabane transmission observed. This was probably due to blocking by one of the other Simbu viruses. ***Incidence***. Not detected. ***Disease***. No disease was observed. ***Features***. Absence of Akabane virus.
1987	***Transmission.*** Sentinel monitoring was limited to Paterson, Singleton and Camden. The Akabane transmission pattern was again unusually late, with seroconversions commencing in May at Paterson and spread to Singleton during May and June. There was no transmission detected at Camden. ***Incidence***. The incidence of Akabane infection was normal (100% at Paterson and Singleton) at the locations where infection was recorded. ***Disease***. No disease was observed. ***Features***. Late transmission of Akabane virus, but at a normal incidence.
1988	***Transmission***. The Akabane transmission pattern was relatively normal, with infection throughout the endemic area. The first recorded infections occurred at about the usual time. Seroconversions were observed between February and April along the North Coast, Central Coast and Hunter regions and in April–May in the upper Hunter and Camden areas.

Akabane in NSW (cont'd)

Incidence. The incidence of Akabane infection was lower than usual on the North Coast, with 20–40% of sentinels seroconverting at Lismore, Grafton and Kempsey but normal, with all or most animals seroconverting, in the Hunter Valley and Gloucester areas. A single animal seroconverted at Camden.

Disease. Not recorded.

Features. A relatively normal Akabane pattern except for the lower incidence on the North Coast.

1989

Transmission. The Akabane transmission pattern was quite unusual in that infection was confined to the far North Coast. The first recorded infections were also much later than usual, and seroconversions were observed only during April and May.

Incidence. The incidence of Akabane infection was also lower than usual, with 20–40% of sentinels seroconverting at Lismore and Grafton and 70–80% at Casino and Coffs Harbour. Infection was not recorded at any other location in NSW.

Disease. Not recorded.

Features. Very limited and late spread of Akabane, probably due to interference from other Simbu viruses, especially Douglas and Tinaroo viruses.

1990

Transmission. Infection of sentinel cattle with Akabane virus occurred throughout the endemic area. There was a distinct north–south pattern of seroconversions, from the northernmost herd at Lismore in February south to Camden in May. There was a low level of transmission in adjoining marginal areas as indicated by the birth of deformed calves.

Incidence. The efficiency of Akabane transmission was reflected in the fact that all sentinel cattle seroconverted in areas where infection was detected.

Disease. Some clinical disease was reported in marginal areas.

Features. Wet summer and autumn conditions.

1991

Transmission. Infection of sentinel cattle occurred throughout the endemic area. Transmission commenced on the North Coast in March and was last detected beyond the endemic region at Nowra in late May–early June.

Incidence. There was a lower rate of seroconversion this year, with a low rate of transmission in adjoining marginal and non-endemic areas.

Disease. Moderate incidence of deformed calves.

1992

Transmission. This was a most unusual year, in that there was virtually no Akabane transmission in the state. The exception was a single focus of activity at Singleton in the mid Hunter Valley. At Singleton, transmission commenced later than usual, with seroconversions only in May. While drought conditions may have played a role in the reduced Akabane transmission, extensive Peaton transmission (see below) may have been a major factor in interfering with Akabane infection.

Incidence. At Singleton, the incidence was low, with only three animals seroconverting.

Disease. No disease was reported.

Features. Severe drought conditions prevailed throughout most of the state; the ensuing winter was unusually cold and dry. It was expected that few, if any, vectors would survive through to the following spring.

Comments. The only Simbu virus to be transmitted extensively was Peaton. Transmission commenced at the usual time at Lismore but was perhaps a little later than usual in the Manning and Hunter valleys.

Akabane in NSW (cont'd)

1993

Transmission. In the north, the onset of transmission was about usual and the pattern of spread typical. The only variation was the failure of transmission westwards beyond the lower parts of the Hunter Valley. Although there was a normal pattern of infection at Paterson, no seroconversions were recorded at Singleton or further inland or south.

Incidence. In the areas where transmission occurred, the incidence was normal, with almost all sentinels seroconverting.

Disease. Interestingly, although there was a high incidence of infection throughout most of the endemic area, little disease was reported, despite the fact that there had been no Akabane activity in these areas the previous year. There were a few confirmed Akabane cases in the Wingham–Gloucester area. The only Akabane transmission in 1992 had been at Singleton, where there was no Akabane this year.

1994

Transmission. The pattern of spread of Akabane virus was relatively normal, with transmission observed throughout the endemic area, to the extreme limits. The southernmost spread was Nowra, but the incidence was very low. The time of onset of transmission was normal on the far North Coast (February), but late on the lower North Coast–Manning regions (first infections in April).

Incidence. The incidence along the coast was quite variable, with the usual very high seroconversion rates (8–10/10) mainly in the Manning, lower Hunter Valley and Camden areas. In the mid and upper Hunter Valley, the incidence was slightly lower but still moderately high. Elsewhere along the coast it was quite variable, tending to be lower than usual (3–6/10 animals seroconverting).

Disease. Small numbers of affected calves were born in the upper Hunter and Camden areas.

Features. Drought or near-drought conditions prevailed for much of the year in many districts but arbovirus activity was not significantly limited.

1995

Transmission. Akabane transmission commenced on the far North Coast at about the normal time, perhaps a little late. The virus spread south as far as the lower Hunter Valley in a normal pattern, although a little later than usual. There was no spread up the Hunter Valley beyond Paterson, or further south.

Incidence. In areas where infection occurred, the incidence was normal, with very high seroconversion rates. At all sites, almost all animals seroconverted with 1–2 months.

Disease. Not observed. Infection was restricted entirely to the endemic area, and was not as expansive as usual.

Features. Drought conditions through most of the state.

1996

Transmission. The pattern was quite abnormal, with no transmission detected.

Incidence. No infection detected.

Disease. Nil.

Features. There were widespread drought conditions early in the season, with a late wet period on the North Coast. Vector distribution was identical to model predictions.

1997

Transmission. Transmission of Akabane on the far North Coast commenced at about the usual time in February. However, subsequent spread was slow, with occasional seroconversions between March and June. Activity was markedly reduced and confined to the established vector areas only on the far North Coast, Manning and lower Hunter Valley regions. The virus did not spread further inland, or further south than Paterson.

Akabane in NSW (cont'd)

Incidence. The incidence was lower than usually observed. Only at Lismore and Casino was there a normal incidence. Elsewhere it was significantly lower, with only 1–3 animals infected.

Disease. Nil.

Features. These results are not unexpected, with low rainfall in most districts. However, Simbu patterns would suggest that there was an effective vector population; Akabane transmission has possibly been affected by earlier transmission of one or more 'other' Simbu viruses.

1998

Transmission. The virus moved to the full extent of its normal range, to the extremities of the coastal strip, south as far as Nowra and up throughout the Hunter Valley. The virus then moved beyond the endemic area into the Merriwa and Mudgee districts in the Central West. Infection occurred along the entire eastern fringe of the Northern Tablelands, and also in the Tenterfield, Armidale, Glen Innes and Inverell districts. Seroconversions were probably a little early, commencing in January. There was a normal pattern of spread, although spread was relatively rapid from the North Coast south to the Hunter Valley. Most animals in these areas seroconverted in February. There was transmission in the upper Hunter, Central West, Northern Tablelands, Camden and upper South Coast areas in March through to May.

Incidence. Within the endemic area, there was a normal, very high incidence, with most sentinels seroconverting. In the areas beyond the endemic margins, there was a moderate seroconversion rate (20–50%).

Disease. Because of this extensive spread, with spillover beyond the endemic area, combined with reduced transmission of the virus in many endemic areas in the previous year or two, there was a significant incidence of disease. In some herds, particularly on the Northern Tablelands, there were moderate to heavy losses of calves, with the usual sequence of both arthrogryposis and hydranencephaly. In some areas, cases were seen in late November, consistent with transmission very late in autumn.

Features. The Akabane results are not unexpected after a period of dry weather, with more favourable rainfall patterns during late summer and autumn, combined with mild autumn temperatures. It is perhaps surprising that there were not even more affected calves within the endemic area, as the incidence of Akabane was so low in the preceding year. Perhaps a measure of immunity is afforded by infection with 'other' Simbu viruses.

1999

Transmission. Although there were early seroconversions on the far North Coast during December 1998, Akabane transmission was not as rapid during the season as may have been expected considering the wet seasonal conditions. Seroconversions were detected in all herds along the coast south to Camden and inland throughout the Hunter Valley as far as Merriwa.

Incidence. Most herds in the endemic area experienced normal rates of infection (with most animals seroconverting), but the time over which seroconversions occurred was longer than usual. There was little 'spillover' beyond the endemic area, probably due to the slow rate of spread, but infection was detected on the eastern fall of the New England Tablelands east of Armidale, at Walcha and Wallangra.

Disease. Because of the extensive spread of the virus in the previous year, only limited numbers of abnormal calves occurred on the Northern Tablelands.

Features. Good seasonal conditions with high rainfall.

Northern Territory

Normal features of Akabane virus transmission in the Northern Territory

Activity is seen in all years in the northerly sites. The time of maximum activity is variable depending on seasonal conditions. In years of high rainfall, activity occurs later in the year, after rainfall has ceased. The period of activity tends to be fairly short, with most animals seroconverting at each site. Disease has not been reported with this virus in the Northern Territory (NT).

Comments relevant to transmission in specific years are as follows.

Akabane in the Northern Territory

Year	Description
1991	***Transmission***. Activity in all northern sites. ***Incidence***. Normal, with most animals seroconverting. ***Disease***. No reported disease. ***Features***. Very good wet season rainfall.
1992	***Transmission***. Activity in all sites south to Newcastle Waters. ***Incidence***. Normal, with most animals seroconverting. ***Disease.*** No reported disease. ***Features***. Very low wet season rainfall.
1993	***Transmission***. Activity in all northern sites. ***Incidence***. Normal, with most animals seroconverting. ***Disease***. No reported disease. ***Features***. Late start and early finish to the wet season. Drought conditions in the Barkly Tablelands and Alice Springs districts.
1994	***Transmission.*** Activity in all northern sites. ***Incidence***. Normal, with most animals seroconverting. ***Disease***. No reported disease. ***Features***. Good rainfall during the wet season over most of the NT.
1995	***Transmission.*** Activity in all northern sites. ***Incidence***. Normal, with most animals seroconverting. ***Disease***. No reported disease. ***Features***. Record rainfall in the northern areas of the NT.
1996	***Transmission***. Activity in all northern sites. ***Incidence***. Normal, with most animals seroconverting. ***Disease***. No reported disease. ***Features***. Late start to the wet season, with poor rainfall until late March–April.

Akabane in the Northern Territory (cont'd)

Year	
1997	***Transmission.*** Activity restricted to Coastal Plains Research Station (CPRS) in the period from January to June. Widespread in the second half of the year. ***Incidence***. Lower than normal in the first half of the year, high in the second half of the year. ***Disease***. No reported disease. ***Features***: Early monsoon, with record rainfall for the season in the north. Drought conditions on the Barkly Tablelands until January.
1998	***Transmission***. Widespread, with activity in all northern sites in each quarter. ***Incidence***. Normal, with most animals seroconverting. ***Disease***. No reported disease. ***Features***. Above-average rainfall in the north and a warm dry season. Drought conditions on the Barkly Tablelands.
1999	***Transmission***. Widespread activity in all northern sites between July and October. ***Incidence***. Normal. High in the most northern sites, lower in the southern Victoria River district ***Disease***. No reported disease. ***Features***. Early start to the wet season. Drought conditions on the Barkly Tablelands.

Queensland

Normal features of Akabane virus transmission in Queensland

Akabane virus transmission occurs seasonally over a wide area of the State. In far northern areas, transmission occurs throughout the year, with a higher incidence in the wetter summer months. In coastal areas, transmission commences in late winter–early spring (Rockhampton) and extends southwards to Maryborough by November and Beaudesert by December. Significant transmission starts in inland areas by December–January (Chinchilla, Dalby, Taroom) and extends west to Roma (February) and on to Charleville and south to border areas by April–May. The onset of winter temperatures and frosts in late May eliminates vector populations, and virus transmission ceases across inland areas.

Comments relevant to transmission in specific years are as follows.

Akabane in Queensland

Year	Description
1994	***Transmission***. No activity in Gulf regions. Transmission in coastal areas ranged from November–December (Ayr, Maryborough) through to April (Innisfail). Extension onto eastern Darling Downs (Dalby) by late summer–early autumn (April–May). No activity detected at Chinchilla, Roma, Condamine, Cunnamulla or Longreach. ***Incidence***. Incidence in coastal areas was 80–100% over a two-month period. Slightly higher incidence (40%) at Dalby. ***Disease***. No reported disease. ***Features***. Second successive year of below-average rainfall over most of the endemic area in eastern Queensland.
1995	***Transmission***. No activity was detected in the Gulf region, but there was midsummer transmission in eastern coastal areas except Maryborough. Extension onto eastern Darling Downs (Dalby) by April. There was no activity in the central highlands or western and southern areas. ***Incidence***. There was a high incidence (80%) in coastal areas and usual incidence (20%) on the Darling Downs. ***Disease***. No reported disease.
1996	***Transmission***. Transmission occurred in September in the Gulf area and in late spring–early summer (November) in eastern coastal areas. No activity was detected in subcoastal or inland areas (Emerald, Clermont, Longreach, Roma, Injune, Chinchilla, Dalby, Cunnamulla or St George). ***Incidence***. Incidence ranged from 25% to 60%, which was slightly lower than normal for endemic coastal areas. ***Disease***. No disease reported.
1997	***Transmission.*** Transmission was restricted to coastal areas throughout the year (for example, in Maryborough, in January, May, November and December), with extension to the eastern Darling Downs in February–March. No activity was detected on the central highlands (Emerald, Clermont), further west (Longreach) or in the south (Cunnamulla, St George). ***Incidence***. Incidence in coastal areas was 30–40% and lower than usual. At Dalby, the incidence of 20% was normal for transmission in that area. ***Disease***. No disease reported.
1998	***Transmission***. In coastal sites north from Rockhampton, transmission commenced in late winter–early spring, but in southern coastal sites (Maryborough, Beaudesert) transmission occurred at the end of summer (February to June). Transmission on the Darling Downs occurred in February–March. ***Incidence***. At coastal sites (Gulf region, Cooktown, Rockhampton, Maryborough, Beaudesert), incidence ranged from 60% to 90%. In inland Central Queensland (Emerald, Clermont), the incidence was 20–30%, but on the Darling Downs it was as low as 10–20%. ***Disease***. No disease reported.

Akabane in Queensland (cont'd)

1999	***Transmission***. Normal pattern during the summer months, with extended transmission into autumn in southern inland areas. Early commencement of transmission in southern areas following winter (Dalby, St George and Warwick). ***Incidence***. There was a high incidence in southern inland areas in early summer (Kingaroy, 80%; Chinchilla, 95%; Warwick, 70%; Charleville 65%). ***Disease***. No disease reported. ***Features***. The late onset of a mild winter saw the early commencement of transmission, with a high incidence in many areas.

Western Australia

Normal features of Akabane virus transmission in Western Australia

Akabane virus seroconversions occur in sentinel cattle in the Kimberley region throughout the wet season (usually November to April). The *Culicoides brevitarsis* vector is endemic in the region, with its activity increasing in the wet season. Akabane disease is infrequently reported in this region, but this is extensive grazing country and there are abundant predators so it is unlikely that reports of Akabane-affected foetuses would be reported with any consistency. The high prevalence in the cattle population in the Kimberley region and the annual cycle of seroconversion in the wet season probably means that most calves are exposed to the virus prior to mating, which should drastically reduce the incidence of Akabane disease.

Comments relevant to transmission in specific years are as follows.

Akabane in Western Australia

Year	Description
1993	***Transmission***. Normal. ***Incidence***. Massive seroconversion (16/16) between March and May 1993. ***Disease***. None reported. ***Features***. Above average in January and February; lower in March but a wet May. ***Comments***. In the extensive grazing country in the Kimberley, and with abundant predators, it is unlikely that reports of Akabane-affected foetuses will be reported with any consistency.
1994	***Transmission***. Normal. ***Incidence***. In Kununurra, seroconversions (2/6) occurred in March and May 1994. In Kalumburu, early seroconversion (July 1993) was probably a carryover from 1992–93, but later seroconversions between November 1993 and June 1994 were consistent with the normal pattern.

Akabane in Western Australia (cont'd)

	Disease. None reported. ***Features***. There was well above average rainfall in the region in December 1993 and February and March 1994. Local climate and geography effects at Kalumburu not directly associated with regional dry season. ***Comments***. In the extensive grazing country in the Kimberley, and with abundant predators, it is unlikely that reports of Akabane-affected foetuses will be reported with any consistency.
1995	***Transmission***. Late in the wet season, corresponding with the very wet April 1995. ***Incidence:*** In Kununurra there was a low level of seroconversion (1/7 available animals). ***Disease***. None reported. ***Features***. Well above average rainfall November to December 1994 and above average February to April 1995. ***Comments***. In the extensive grazing country in the Kimberley, and with abundant predators, it is unlikely that reports of Akabane-affected foetuses will be reported with any consistency.
1996	***Transmission***. Normal. ***Incidence***. In Kununurra, there was massive seroconversion (11/13) in March 1996, with a single preceding seroconversion (1/14) in November 1995. In Kalumburu, there was moderate seroconversion for the year (4/13). ***Disease***. None reported. ***Features***. January 1996 was fairly dry, but there was otherwise an average wet season. ***Comments***. In the extensive grazing country in the Kimberley, and with abundant predators, it is unlikely that reports of Akabane-affected foetuses will be reported with any consistency.
1997	***Transmission***. In Kununurra, transmission in August 1996 relates to the heavy period of Akabane activity in 1995–96. The rest of the year showed a normal transmission pattern at both Kununurra and Kalumburu. ***Incidence***. In Kununurra the incidence was moderate for the year (7/15); at Kalumburu, it was low (1/12). ***Disease***. None reported. ***Features***. There was above-average rainfall in January 1997 and a fairly normal wet February, preceding a fairly dry March and April. ***Comments***. In the extensive grazing country in the Kimberley, and with abundant predators, it is unlikely that reports of Akabane-affected foetuses will be reported with any consistency.
1998	***Transmission***. Normal at Kununurra. At Kalumburu, seroconversion in August 1997 was not related to regional rainfall activity. Seroconversion in February 1998 was consistent with the normal pattern. ***Incidence***. In Kununurra, only three seronegative sentinels were available, due to the Akabane activity of the previous year; 1/3 seroconverted in January 1998. In Kalumburu, the incidence was moderate (4/12 for the year). ***Disease***. None reported. ***Features***. Fairly average wet season. ***Comments***. In the extensive grazing country in the Kimberley, and with abundant predators, it is unlikely that reports of Akabane-affected foetuses will be reported with any consistency.

Bluetongue virus

New South Wales

Normal features of bluetongue virus transmission in New South Wales

Bluetongue virus transmission in New South Wales (NSW) is confined to the coastal regions of the State, from the Queensland border in the north, extending just south beyond the Hunter Valley. Its potential distribution is the same as Akabane virus, as these viruses have a common vector, *Culicoides brevitarsis*. However, bluetongue viruses appear to be spread much less efficiently than Akabane virus: they are rarely spread as expansively, and transmission commences later in the season. Bluetongue viruses may be spread inland towards the eastern fall of the Great Dividing Range, with the western limit being defined by altitude as cooler temperatures influence the spread of *Culicoides* vectors. Spread beyond these limits, to either the west or south, is rare. Unlike Akabane, infection has never been recorded in herds on the Northern or Central Tablelands of the range. Southern spread beyond the limit of the defined endemic vector area occurs only under extremely favourable conditions, particularly when there are mild temperatures and, to a lesser extent, relatively high rainfall. Spread beyond the endemic region has perhaps been associated with windborne dispersal of vectors. Bluetongue viruses have always been confined to coastal regions where sheep are uncommon and have not been spread in commercial sheepraising districts of NSW. Virus transmission occurs only during the warmer summer and autumn months. In the far northern districts, infections usually occur in late February–March, or with very favourable conditions in January. On the lower North Coast and in the Hunter–Manning regions, transmission commences in March–April. Transmission may continue in the endemic region until late May–early June, but is not observed during the colder months of winter and spring. Within the *Culicoides brevitarsis* endemic coastal region, transmission does not occur every year and spread is not uniform throughout the vector region. Transmission is more common in the far northern coastal areas and in the Manning region. When infection is detected in the Hunter Valley, it is not commonly detected west of Singleton in the inland upper Hunter Valley. When infection is observed, the incidence varies from year to year and between locations. In some years there can be a moderate to high incidence, with a majority (> 70–90%) of sentinel animals seroconverting. In other years, even in areas where transmission occurs more frequently, seroconversion rates may be much lower, with 30–50% of cattle becoming infected. Only serotypes 1 and 21 have been detected in NSW. Serotype 1 predominates; type 21 is detected infrequently. Natural infection of sheep is relatively uncommon, but even under experimental conditions NSW strains of type 1 appear to be nonpathogenic. Comments relevant to transmission in specific years are as follows.

Bluetongue in New South Wales

Year	Description
1978-81	First years of prospective monitoring for bluetongue in NSW (Littlejohns and Burton 1988).
1986	Monitoring occurred at Paterson, Singleton and Camden. No transmission was observed.
1987	Monitoring occurred at Paterson, Singleton and Camden. No transmission was observed.
1988	***Transmission.*** Probably the first year for some time when there was quite widespread transmission of this virus. Infection was observed along the North Coast and in the Hunter–Manning regions. There was a break in transmission on the mid-North Coast, with no seroconversions at Kempsey. The onset of infection occurred at about the time expected, but transmission occurred over a prolonged period, from February through to June. ***Incidence***. A very high incidence was observed on the far North Coast and in the Hunter Valley (> 90% at Lismore, Paterson and Singleton), with a moderate incidence (50–60%) at Grafton and Gloucester. ***Features***. This was the probably the first occasion when such a high incidence of bluetongue virus infection was documented in the Hunter Valley. A low level of infection (about 25%) was detected in sheep at Paterson. Infection was predominantly due to type 1, but there was probably a low incidence of type 21 infections in most districts. Eight isolates of type 1 were obtained from sentinel cattle in the Hunter Valley.
1989	***Transmission***. An exceptional year, indicated by the widespread transmission of bluetongue virus, probably emanating from a focus on the lower North Coast–Manning region. This may have been a continuation of spread from the previous year, perhaps due to overwintering of the virus in a central coastal location. As well as the unusual pattern and the extent of spread (south as far as Bodalla), the first recorded seroconversions were unusually early (January at Taree, compared to March–April that far south). ***Incidence***. A very high incidence was observed on the lower North Coast, Manning region, Hunter Valley and Camden district. All sentinels seroconverted at Taree, Gloucester, Scone and Camden. When larger numbers of cattle and sheep were bled, these trends were confirmed (see comments below). ***Features***. This was the first occasion when bluetongue virus transmission was documented over such a large area, with a high incidence and extending so far south. In some districts there was apparently a saturation of the cattle population with significant 'spillover' to sheep). ***Comments***. Intensive sampling at Elizabeth Macarthur Agricultural Institute (EMAI) Camden revealed the following incidence: sentinels: 10/10; dairy cattle: 145/157; sheep: rams 28/52; ewes 3/30; wethers 5/32 (running with rams); goats: 22/50; At Paterson: sentinels 6/10; sheep 1/20 (1988 sentinels 10/10; sheep 7/26); At Singleton: sentinels 4/10; sheep 0/20; At Berry/Nowra: 80% of bulls and approx 40% of cows both based on >100 samples. Twelve isolates of virus were obtained from sentinel cattle. Only type 1 transmitted.

Bluetongue in New South Wales (cont'd)

1990

Transmission. There were no bluetongue seroconversions detected in cattle or sheep.

Features. Wet summer and autumn conditions.

1991

Transmission. There were no bluetongue seroconversions detected in cattle or sheep.

1992

Transmission. No activity was detected anywhere in the State.

1993

Transmission. Infection was limited to the far North Coast (Grafton and further north), with a second isolated focus on the mid-North Coast at Taree. Seroconversions were probably a little later than usual: the first was observed in March at Lismore, with a few seroconversions in April and May and then more infections at Grafton and Taree in June (May transmission).

Incidence. The incidence was generally lower than usual, with 3–4 animals seroconverting at all locations except Grafton, where there were seven animals infected.

Features. Again there was an apparently independent focus at Taree.

Comments. Four isolates of BLU 1 were obtained.

1994

Transmission. An unusual pattern emerged in that, while infection was widespread along the coast of NSW, it appeared to be quite distinct from movement from interstate. Transmission was also detected further south and west than usual. The first seroconversions occurred at Coffs Harbour in January; there appeared to be a progressive southern movement subsequently. Infection was detected as far west as Scone and south as Camden, where the seroconversions were first observed in June. Interestingly, there was a single (very late) seroconversion at Lismore in June, with no infection at Casino or Grafton during the year.

Incidence. From Coffs Harbour south, at all coastal locations and throughout the Hunter Valley, the incidence was quite high (6–10/10). At Camden, two animals seroconverted, but infection commenced very late in the year, as it did at Lismore (see above).

Features. A major feature was the lack of infection on the far North Coast, perhaps further evidence of an endemic focus, with southern movement from the mid-North Coast. (This feature has been observed in at least one other year, with movement from Kempsey.) Another interesting observation was the relatively high infection rate in sheep in a small flock at Gloucester (63%, 14/22), with no disease observed. Further reflecting the vector activity around these sheep was the high incidence of Akabane (> 95%, 21/22).

1995

Transmission. Not detected.

1996

Transmission. Transmission of bluetongue virus was perhaps slightly late and was confined entirely to the far North Coast, adjacent to the Queensland border. Surprisingly, seroconversions were recorded only at Lismore, and none at the nearby site at Casino.

Incidence. There was only a moderate incidence of infection, with five of the sentinels seroconverting.

Bluetongue in New South Wales (cont'd)

1997	***Transmission.*** Transmission of bluetongue virus was widespread on the North Coast and Central Coast but did not spread further south than Taree. The first seroconversions were probably earlier than usual, commencing in January at Casino and continuing through to June. Activity at Taree was again detected earlier than at the more northern sites of Coffs Harbour and Kempsey. ***Incidence***. Relatively normal at all sites where infection occurred, though perhaps a little lower on the far North Coast (7–8 positives compared to all 10 at Kempsey and Taree). ***Features:*** Infection was limited to serotype 1. Seventeen isolates of bluetongue virus were obtained from sentinel cattle bloods.
1998	***Transmission***. Transmission of bluetongue virus was recorded at sites along the North and mid-North Coast, south to Taree. Spread was relatively slow, commencing in March at Lismore and Casino and continuing each month through to July, when the last seroconversion was recorded at Taree. There was limited infection at Coffs Harbour. This segregation of transmission between the far North Coast and lower North Coast–Manning region has been observed previously. In some years, there is a distinct pattern of northwards movement from the Taree–Kempsey areas ***Incidence***. The incidence was moderate, with a usual range of 5–8 animals seroconverting. The exception was Coffs Harbour, where there was a single seroconversion. ***Features***. Infection was limited to serotype 21. This was the first occasion for about 10 years that type 21 was found in New South Wales; it had not previously been spread as extensively. Eleven isolates of type 21 virus were obtained from sentinel cattle bloods. Genetic analysis showed two distinct clusters of viruses: those from the far North Coast and those in the Hastings–Manning region. These results suggest that there is an endemic focus of infection in central coastal NSW, with either a second focus in the far north of the State or a gene population that is shared in the north with Queensland.
1999	***Transmission***. During the season, seroconversions were limited to three herds on the far North Coast, at Lismore, Casino and Coffs Harbour. ***Incidence***. Seroconversion of 50–70% of animals was observed In these herds. ***Features***. A number of isolates of type 1 were obtained.

Northern Territory

Normal features of bluetongue virus transmission in the Northern Territory

In the Northern Territory (NT), activity normally occurs each year at the three most northern sites (Berrimah, Coastal Plains and Douglas Daly). The serotypes active each year vary; BLU 1 is the most common. Activity occurs in some, but not all, years at Katherine and Victoria River. Activity normally occurs between January and May, but at Victoria River Research Station (VRRS) activity has been seen in August–September. Prolonged activity extending into August and September occurs in some years at Coastal Plains Research Station (CPRS). The incidence is normally high at the most northerly sites and lower at the inland sites.

Comments relevant to transmission in specific years are as follows.

Bluetongue in the Northern Territory

Year	Description
1991	***Transmission***. Abnormal. Activity was confined to CPRS, where BLU 3 was isolated. ***Incidence***. Abnormal. At CPRS, only 2/20 animals were infected. There were no seroconversions at any other site. ***Features***. Very good wet season rainfall.
1992	***Transmission***. Normal. Activity restricted to the northern coastal areas and Douglas Daly Research Farm (DDRF). BLU 16 was isolated at CPRS. ***Incidence***. Normal. ***Features***. Very low wet season rainfall.
1993	***Transmission***. Normal. BLU 1 activity was confined to CPRS, BARC and DDRF. ***Incidence***. Normal. ***Features***. Late start and early finish to the wet season. Drought conditions in the Barkly Tablelands and Alice Springs districts.
1994	***Transmission***. Normal. BLU 1 and BLU 21 were isolated at CPRS. ***Incidence***. Normal. ***Features***. Good rainfall during the wet season over most of the NT.
1995	***Transmission***. The first isolations of the wet season occurred at CPRS in October 1994, when BLU 21 was isolated. Activity was restricted to CPRS, BARC, DDRF and Katherine Research Station (KRS). BLU 21 was active at all sites, with BLU 20 at CPRS, DDRF and KRS. ***Incidence***. Normal. There was a very high level of virus isolation at CPRS. ***Features***. Record rainfall in the northern areas of the NT.
1996	***Transmission***. Normal. BLU 20 was active at CPRS; there was serological evidence of BLU 21 at BARC and BLU 21 and BLU 1 at DDRF. ***Incidence***. Normal. ***Features***. There was a late start to the wet season, with poor rainfall until late March–April.
1997	***Transmission***. More extensive than usual, with activity in all northern sites. BLU 1 and BLU 20 were both active. ***Incidence***. Above-average numbers of viruses were isolated at CPRS. ***Features***. Early monsoon with record rainfall in the north. Drought conditions on the Barkly Tablelands until January.

Bluetongue in the Northern Territory (cont'd)

1998	***Transmission***. There was no activity at BARC. At VRRS, there was BLU 21 activity in June. BLU 1 was active at CPRS and DDRF. ***Incidence***. Abnormal. Virus activity was low at CPRS. ***Features***. Above-average rainfall in the north and a warm dry season. Drought conditions on the Barkly Tablelands.
1999	***Transmission***. BLU 1 and BLU 20 were active at BARC, CPRS and DDRF. BLU 1 was active at KRS and VRRS. ***Incidence***. All animals seroconverted at each site except VRRS, where only 1/20 seroconverted. ***Features***. Early start to the wet season. Drought conditions on the Barkly Tablelands.

Queensland

Normal features of bluetongue virus transmission in Queensland

Bluetongue virus transmission occurs seasonally over a wide area of northern and eastern Queensland. On Cape York Peninsula, in the Gulf area and on the north tropical coast, transmission commences in late spring (October–November) and continues throughout the wet summer months. Incidence in these areas drops dramatically with the onset of the dry season and cooler winter temperatures from June to October. In central Queensland, transmission commences on the coast (Rockhampton) and adjacent highlands (Clermont, Emerald) in December–January. Transmission extends southwards, reaching Maryborough by February–March and the southern coast (Beaudesert) by March. In inland areas, transmission commences on the northern and eastern Darling Downs by March–April and slowly extends southward to border areas by May in favourable years. With the onset of cooler temperatures and frosts in late May, vector populations disappear from inland southern Queensland and virus transmission ceases.

Comments relevant to transmission in specific years are as follows.

Bluetongue in Queensland

Year	Description
1994	***Transmission.*** Both serotype 1 (Maryborough, Dalby) and serotype 21 (Innisfail) were detected. In coastal areas, the activity was in late summer–early autumn (April–June). There was activity on the Darling Downs, with the same serotype as Maryborough but extending into June. ***Incidence***. Normal. ***Features***. Most of the State recorded below-average rainfall for the year, with drought conditions on the eastern Darling Downs.

Bluetongue in Queensland (cont'd)

1995

Transmission. Both serotypes 1 and 21 were detected, with activity restricted to coastal areas in late summer and early autumn (April–June). Type 21 activity occurred only at Weipa and extended into August and September. There was no activity in subcoastal or marginal areas.

Incidence. The 10% seroconversion at Maryborough was the lowest since monitoring began at the site. The lack of activity in subcoastal areas may reflect low activity in coastal areas.

Features. Below-average annual rainfall throughout most of the endemic and adjacent areas.

1996

Transmission. Transmission was detected only in coastal areas from Weipa to Maryborough. Both serotypes 1 and 21 were detected at Maryborough.

Incidence. Low (20%) at Weipa but high (100%) at Maryborough. The year was unusual in that no seroconversions were detected at Cooktown.

Features. Average to below-average annual rainfall throughout most of the endemic area and adjacent marginal areas.

1997

Transmission. Transmission was detected in coastal areas from Weipa to Maryborough in the late summer–autumn (April–May). Activity was detected on the Darling Downs (Dalby) in March and April after an absence of two years.

Incidence. Normal.

Features. Low summer and winter rainfall in central and southern areas saw activity largely restricted to coastal areas.

1998

Transmission. Transmission of both serotypes 1 and 21 occurred widely in the endemic zone, with unusual activity in northern coastal areas (Rockhampton, Townsville and Burketown areas) extending into spring and early summer (August, September and November). In coastal and subcoastal areas of southern and central Queensland, transmission occurred at the usual time of mid to late summer (January to May).

Incidence. The incidence of infection was abnormally high in Townsville, there having been no cases for over two decades.

Features. There was exceptional rainfall in the Townsville area throughout the year, with an annual total of 2660 mm, which is 2.5 times the long-term annual figure. Heavy early monsoonal activity was experienced over the southeastern Gulf of Carpentaria.

1999

Transmission. Normal pattern in northern and central areas of the State. Activity in southern coastal areas and the Darling Downs commenced earlier (December–January) than usual and extended into autumn.

Incidence. There was a high incidence of infection at some sites (Maryborough, 100%; Beaudesert, 80%; Goondiwindi, 40%).

Disease. No.

Western Australia

Normal features of bluetongue virus transmission in Western Australia

Bluetongue virus seroconversions can occur in sentinel cattle in the Kimberley region throughout the wet season (usually November–April). This appears to depend on the rainfall pattern and its influence on numbers of the *Culicoides brevitarsis* vector. The local rainfall patterns at different monitoring centres in the Kimberley region seem to be more important than regional rainfall patterns, as it is common to get quite different seasonal seroconversion patterns at Kalumburu and Kununurra. Some local rainfall–vector breeding patterns at these monitoring sites have also been associated with late seroconversions (June to September) in sentinel cattle in some years. No bluetongue seroconversions are detected in sentinel cattle south of this region. The serotype specificity for seroconverting sentinel cattle in the Kimberley region generally matches that for sentinel cattle in the western part of the Northern Territory.

Comments relevant to transmission in specific years are as follows.

Bluetongue in Western Australia

Year	Description
1990	***Transmission***. Relatively late for Kununurra. ***Incidence***. Only 1/14. ***Features***. The 1989–90 wet season lacked heavy rain, but extended through April and May 1990 and probably contributed to the late seroconversion.
1991	***Transmission***. Lower than normal considering the fairly average wet season. ***Incidence***. One of nine animals available in February. ***Features***. There was slightly higher than average rainfall in February.
1992	***Transmission***. The onset time in December was normal but seroconversions occurred from December through to March. ***Incidence***. Nine out of 16 animals seroconverted. ***Features***. The wet season had below-average rainfall but extended through to a fairly wet April.
1993	***Transmission***. Relatively late in the season (March, May) at Kununurra. ***Incidence***. Only 2/17. ***Features***. Rainfall was above average in January and February; it was lower in March but there was a wet May.
1994	***Transmission***. At Kununurra, there were major seroconversions between November and January. At Kalumburu, there were seroconversions in November and a major seroconversion in April. ***Incidence***. In Kununurra, 14/15 seroconverted. In Kalumburu, 3/11 seroconverted in November 1993 and 7/7 to BT 1 in April 1994, but seroconversions still occurred in replacement cattle up to July 1994.

Bluetongue in Western Australia (cont'd)

Features. The region had well above average rainfall in December 1993, and February and March 1994. Local climate and geography effects at Kalumburu not directly associated with regional dry season.

Comments. Vector numbers did not reflect the heavy seroconversions at Kununurra in this year. Perhaps the weather interfered with light traps.

1995

Transmission. At Kununurra, there was a major seroconversion in January 1995. At Kalumburu, there was a prolonged period of seroconversion from January to May.

Incidence. In Kununurra, 6/14; in Kalumburu, 5/7.

Features. Rainfall was well above average in November–December 1994 and above average from February to April 1995.

1996

Transmission. None for Kununurra. At Kalumburu, there was some activity (BLU 21) in November 1995 and a major seroconversion between May and June 1996 (BLU 20 and 21).

Incidence. In Kalumburu, 5/10.

Features. January 1996 was fairly dry but the wet season was otherwise average.

Comments. At Kununurra there was no activity despite reasonable *C. brevitarsis* numbers from January to March.

1997

Transmission. There was an unusual late seroconversion at Kununurra between June and August 1996. At Kalumburu there were seroconversions in August and September 1996.

Incidence. The incidence was 1/8 at Kununurra, 3/5 at Kalumburu.

Features. Preceded by a long wet season, but the region had been dry at the time of seroconversions.

Comments. *C. brevitarsis* numbers were unusually high in July 1996 at Kununurra and relatively high in July and August at Kalumburu.

1998

Transmission. An unusual seroconversion pattern was again seen at Kununurra in August and September 1997; there was more normal seroconversion at Kununurra in November 1997 and January and March 1998. There was no activity at Kalumburu.

Incidence. The incidence was 4/11 available in August and September 1997 and 4/7 in November 1997 and January 1998. There were 3 seroconversions in 9 replacement sentinels in March.

Features. There were no general regional rainfalls to correlate with the August–September seroconversions; there was a fairly average wet season.

Comments. There was persistent *C. brevitarsis* activity in the July, August and September 1997 light traps; there were relatively large numbers from November to March 1998.

Bovine ephemeral fever virus

New South Wales

Normal features of bovine ephemeral fever virus transmission in NSW

Infection of cattle with bovine ephemeral fever virus (BEF virus) is most frequently seen along the coastal plains of the Central to far North Coast and in the Hunter Valley. There tends to be a high incidence of infection every second or third year, with sporadic (or no) infections detected in intervening years. A higher incidence is often associated with periods of very high rainfall, reflecting the spread of this virus by mosquitoes. In sentinel cattle, the incidence of seroconversion is usually moderate to high (60–90%) in one year, followed by 1–2 years of low incidence (< 20%). Infection is usually first observed in January–February in the far north of the state and February–March in the central coastal districts of the state, reflecting periods of high temperature, summer rainfall and large mosquito populations. In some years early transmission is observed in late December–early January. Spread can continue for a prolonged period until very cold winter conditions are encountered. Within the coastal region where ephemeral fever virus is endemic, disease outbreaks are observed every 2–3 years as the proportion of susceptible stock increases. Disease usually occurs in younger cattle (under 3–4 years) and a small proportion of very old animals. Disease is not common in young calves. Occasionally outbreaks of disease occur in inland parts of the state, mostly in the northwest, in association with periods of high rainfall, reflecting the wider distribution of the mosquito vector.

Comments relevant to transmission in specific years are as follows.

BEF in New South Wales

Year	Description
1975	First endemic focus of BEF documented in NSW (St George et al 1977). Prior to 1975, BEF was only seen in NSW as it was covered by a north–south wave of infection that was observed in the early major epidemics.
1972–81	Pattern of transmission changes from sweeping epidemics to cyclic epidemic waves and endemic foci in NSW (Kirkland 1982).
1981–85	Endemic transmission continues in NSW (Uren et al 1987).
1986	***Transmission***. Sentinel monitoring occurred at Paterson, Singleton and Camden. No transmission was observed. Seroconversions were detected at Paterson in March and at Singleton in May and June. Not detected at Camden. ***Incidence***. High at Paterson (100%), moderate at Singleton. ***Disease***. There were sporadic cases in the Hunter Valley and Gloucester in December. Epidemics in these areas (Hunter Valley from the coast to Singleton) commenced in late February and peaked in March–April. There were also cases on the far North Coast (Casino) in February, but none were reported on the mid North Coast (Taree, Wingham and Kempsey). There was a late spread to the upper Hunter Valley and Hawkesbury districts. ***Features***. Significant epidemic in the Hunter region.

BEF in New South Wales (cont'd)

1987	***Transmission***. Monitoring occurred at Paterson, Singleton and Camden. No transmission was observed. ***Incidence***. Not detected. ***Disease***. Not observed.
1988	***Transmission***. Transmission occurred as two apparently separate foci, with extensive infection in the Gloucester and lower Hunter Valley regions, and later on the far North Coast. Seroconversions were detected first at Gloucester in early February and at Paterson in April. A single seroconversion was detected at Lismore in April, with transmission continuing through to late June. No seroconversions were detected at Kempsey, although sporadic clinical cases occurred in the district. Infection was not observed south of the Hunter Valley. ***Incidence***. The incidence was high at Gloucester (90%), moderate at Paterson and Lismore (50%) and low (20–30%) elsewhere. ***Disease***. An outbreak occurred in the Gloucester district. There were sporadic cases throughout the lower to mid Hunter Valley and Dungog areas. Sporadic clinical cases were also observed on the far North Coast. ***Features***. As has been observed previously (since the mid-1970s), an outbreak commenced in central coastal NSW independently of any wave of infection moving from the north.
1989	***Transmission***. Transmission was sporadic and confined to the far North Coast and lower Hunter Valley regions. Seroconversions were observed over a wide time span (January–June), though only a few animals were infected. ***Incidence***. The incidence was low to moderate, with single seroconversions at Grafton and Paterson and 7–8 at Casino and Coffs Harbour. ***Disease***. Sporadic clinical cases were observed on the North Coast, commencing in January.
1990	***Transmission***. There were no seroconversions during the monitoring period. ***Disease***. None reported. ***Features***. Wet summer and autumn conditions.
1991	***Transmission***. Transmission commenced in Gloucester and spread in a radiating pattern to the north, south and southwest. There were no seroconversions north of Grafton, but transmission was observed south to Camden ***Incidence***. Up to 40% incidence of disease in some herds. ***Disease***. Widespread clinical disease in the Hunter–Manning region and South Coast, with sporadic cases in the west of the state. ***Features***. Mild winter.
1992	***Transmission***. Seroconversions were observed a little later than usual, first in March in the Hunter Valley at Paterson and in the Hawkesbury–Nepean areas, and then radiating out along the coastal strip to the north and south. There was eventually infection in all herds from Lismore to Nowra. Infection was not detected in sentinels further inland than Paterson. ***Incidence***. There was a moderate to high incidence (4–9/10) in sentinels in the Hunter, Manning and Nepean valleys; north of Taree, the incidence was low (1–2 seroconversions), except for a single focus of high incidence (10/10) at Coffs Harbour. Also see below for inland NSW.

BEF in New South Wales (cont'd)

Disease. Clinical cases were observed from late February to early March, with significant outbreaks in the lower Hunter, Manning, Hawkesbury and Nowra areas. Sporadic cases, many confirmed serologically, occurred in the northwest of the state in the Tamworth, Coonamble, Narrabri, Moree and Walgett areas. In these inland areas, clinical cases were found in close association with groundwater reserves and streams.

1993

Transmission. No transmission was recorded in sentinels anywhere in the state.

1994

Transmission. There was extensive ephemeral fever virus transmission, especially along the coast, with two separate foci of infection commencing in the Hunter–Manning region and on the far North Coast. The other notable feature was the spread up the Hunter Valley beyond Scone and west to Dubbo. There were no seroconversions south of the Hunter Valley. The onset of transmission was about normal, with seroconversions occurring first in February and continuing through to June.

Incidence. In most districts the incidence was only moderate, with 2–5 animals seroconverting. The exception was at Paterson in the Hunter Valley, where all sentinels seroconverted by March.

Disease. The disease was particularly severe at Paterson in the Hunter Valley, with 9/10 sentinels very sick and all seroconverting within one month. Although no sentinels seroconverted south of the Hunter Valley, clinical cases were observed south as far as Mittagong.

Features. The distinct foci of infection were a feature. Also of note were the apparent spread of the virus up the Hunter Valley well into the central west, and the close association between surface water and the occurrence of clinical cases. The latter two observations provide further evidence for a mosquito vector.

1995

Transmission. No infection detected in sentinels anywhere in the state. However, there was a small focus of infection in the Narrabri area (there were no sentinels in this area).

Incidence. Low to moderate in the Wee Waa area near Narrabri.

Disease. An isolated small focus involving a few properties.

Features. Very isolated focus, perhaps in conjunction with spread down inland rivers from Queensland.

1996

Transmission. Ephemeral fever was widespread in NSW. The time of the commencement of transmission was normal, perhaps a little late in the north. Transmission in coastal areas was probably a little longer than usual, with seroconversions observed over 3–4 months. The most distinctive feature was the widespread transmission of the virus in inland areas, particularly the extensive spread in the northwest of the state. Infection was observed in all districts in the northwest (especially Narrabri and Moree) and the central west (Parkes) and spread south as far as Wagga Wagga. Limited cases were even recorded on the Northern Tablelands at Armidale. (Cases were also confirmed on a number of properties in northern Victoria in the Strathbogie ranges.)

Incidence. Seroconversions were only recorded in coastal and lower Hunter Valley sentinels. Herds elsewhere in the State remained seronegative. There were, however, numerous laboratory confirmations of clinical cases throughout the state. The incidence in sentinels on the coast ranged from moderate to high (4–8 seroconversions).

Disease. Clinical cases were observed (and confirmed: see above) in all areas where infection was reported. In this instance there was also a significant occurrence of disease in sentinels in the North Coast and lower Hunter locations.

BEF in New South Wales (cont'd)

	Features. Although there were drought conditions for most of the year, there was good rainfall during the autumn — obviously enough to support extensive mosquito activity, but only limited midge populations.
1997	***Transmission***. Ephemeral fever was widespread on the coast and in the northwest of the State. Spread along the coast extended south to the Hunter Valley and inland to Singleton. Transmission of the virus in these inland regions followed a pattern observed in Queensland and South Australia, following extensive flooding in the centre. Transmission on the coast was relatively late, being detected first in April and extending through to June. ***Incidence***. Seroconversions and clinical cases were generally sporadic. The highest incidence was at Narrabri (5/10); most locations recorded seroconversion in 1–3 animals. ***Disease***. There were moderate numbers of cases in the northwest of the State from Narrabri to Bourke. ***Features***. Cases occurred over a wide area in inland NSW, Queensland and South Australia following flooding, but the incidence was not high.
1998	***Transmission***. No seroconversions recorded. ***Disease***. Occasional sporadic cases of ephemeral fever-like disease were reported, and investigated, but none was confirmed as ephemeral fever.
1999	***Transmission***. Ephemeral fever transmission was observed, with seroconversions detected on the far and mid North Coast during January and February and continuing in the Hunter Valley during March to May, with a low incidence of infection west as far as Scone. ***Incidence***. Low to moderate. ***Disease***. No clinical cases were reported.

Northern Territory

Normal features of bovine ephemeral fever virus transmission in the Northern Territory

BEF activity occurs each year in the northerly sites. In years of above-average rainfall, activity may extend as far south as the Alice Springs area. Seroconversions tend to occur over a number of months and can happen at any time of the year. Most animals are infected in the northern sites, but the incidence is low when infection occurs in the southern herds. Clinical disease is usually seen each year in the north.

Comments relevant to transmission in specific years are as follows.

BEF in the Northern Territory

Year	Description
1991	***Transmission***. Normal. Activity at all northern sites. ***Incidence***. Normal, with most animals seroconverting. ***Disease***. Clinical disease reported from the Darwin and Katherine districts. ***Features***. Very good wet season rainfall.
1992	***Transmission***. Normal. Activity at all sites south to Newcastle Waters. ***Incidence***. Normal. ***Disease***. None reported. ***Features***. Very low wet season rainfall.
1993	***Transmission***. Normal. Activity at all sites south to Newcastle Waters. ***Incidence***. Normal. ***Disease***. None reported. ***Features***. Late start and early finish to the wet season. Drought conditions in the Barkly Tablelands and Alice Springs districts.
1994	***Transmission***. Normal. Activity at all sites south to Rockhampton Downs. ***Incidence***. Normal. ***Disease***. Extensive clinical disease on the Barkly Tablelands and Gulf region. ***Features***. Good rainfall during the wet season over most of the NT.
1995	***Transmission***. Activity at all sites south to Rockhampton Downs. ***Incidence***: Normal. ***Disease***. Clinical cases were reported in export cattle in Darwin and a number of subcoastal properties. Some were confirmed by virus isolation or rising titres. ***Features***. Record rainfall in the northern areas of the NT.
1996	***Transmission***. Activity was restricted to CPRS, BARC, KRS and DDRF between October and March. ***Incidence***. Normal. ***Disease***. Clinical cases were reported at all sentinel sites. ***Features***. Late start to the wet season with poor rainfall until late March–April.
1997	***Transmission***. Activity extended south to the Alice Springs area. ***Incidence***. Very high incidence at most sites. ***Disease***. Extensive reports from all areas of the NT, including south of Alice Springs. Interruptions to live exports in Darwin. Many cases confirmed by virus isolation or polymerase chain reaction. ***Features***. Early monsoon, with record wet season rainfall. Drought conditions on the Barkly Tablelands until January.
1998	***Transmission***. Widespread in northern areas. ***Incidence***. Normal. ***Disease***. Clinical disease reports in northern areas. Some were confirmed by virus isolation or rising titres.

BEF in the Northern Territory (cont'd)

	Features. Above-average rainfall in the north and a warm dry season. Drought conditions on the Barkly Tablelands.
1999	***Transmission***. Widespread in the northern sites from April to December, extending to Alice Springs in April. ***Incidence***. Normal; 1/20 seroconverted at Alice Springs. ***Disease***. Numerous cases seen in export cattle on agistment at CPRS. Disease confirmed on a property to the northwest of Alice Springs. ***Features***. Early start to the wet season. Drought conditions on the Barkly Tablelands.

Queensland

Normal features of bovine ephemeral fever virus transmission in Queensland

Transmission of BEF virus occurs over a wide area of the State. In northern and central areas, transmission occurs throughout the year, but in inland and southern areas it is confined to the summer months. Transmission starts in July–August in coastal areas of the north (Townsville), by September in central areas (Rockhampton), November in Maryborough and January in the far south (Beaudesert). On the Darling Downs, transmission starts by January and continues until May; it extends west to Charleville and south to the NSW border (Warwick, Goondiwindi, St George). Transmission ends in these areas with the onset of winter temperatures and frosts. Far western areas remain free.

Comments relevant to transmission in specific years are as follows.

BEF in Queensland

Year	Description
1993	***Transmission***. Few sites were used for BEF surveillance in 1994. Activity was only detected at Maryborough from December to March; there was no activity at Bamaga, Rockhampton or Millaroo (Swans Lagoon). The only inland site monitored, Roma, was negative. ***Incidence***. There was a 30% incidence at Maryborough, but insufficient data to enable meaningful comment. ***Disease***. No data recorded. ***Features***. Most of the endemic area recorded below-average rainfall for the year. The only area of the State recording average annual rainfall was the Gulf and Peninsula region.
1994	***Transmission***. Transmission occurred from November to April in the Gulf (Normanton, Burketown); slightly later (June to August) in eastern coastal areas (Millaroo); and in summer–autumn in southern inland areas (Dalby, Chinchilla). Transmission also occurred at Cunnamulla, but seasonal data are not available because there were only two bleeds in the year. No activity was detected at Rockhampton, Maryborough, Longreach or Roma–Injune.

BEF in Queensland (cont'd)

	Incidence. The incidence was 20–40% except at Cunnamulla, where it was 10%. There were no areas of high incidence. ***Disease***. No data recorded. ***Features***. Drought conditions throughout many areas, with a second successive year of below-average rainfall.
1995	***Transmission***. In far northern areas, transmission occurred from January to July; in southern coastal areas, it occurred from April to July. There was a patchy distribution, with no transmission detected at Rockhampton or Millaroo (Swans Lagoon) or at any inland sites (Longreach, Roma, Dalby, St George, Cunnamulla). ***Incidence***. The incidence was high (100%) at Cooktown, lower in the Gulf (Burketown 40%) and southern coastal areas (Maryborough 30%). ***Disease***. No data available.
1996	***Transmission***. Transmission was widespread throughout the State but not uniform. Northern coastal sites recorded high seroconversion rates (80%), but no activity was detected in southern coastal areas. There was a similar patchy distribution in inland sites, with 100% infection at Emerald, no activity at Dalby, 80% infection nearby at Chinchilla, 20% at St George and 100% at Cunnamulla. In southern areas, infection occurred during summer and autumn (January to May); in far northern areas (Cooktown, Normanton), transmission occurred from June to September. ***Incidence***. See above. ***Disease***. Usual reports of clinical disease.
1997	***Transmission***. There were insufficient data recorded to comment on a State-wide basis. On the Darling Downs and Maranoa, there were seroconversions from January to June. ***Incidence***. Incidence ranged from 20% at Dalby and St George to 100% at Cunnamulla. ***Disease***. Clinical disease was reported but no confirmatory diagnoses were sought.
1998	***Transmission***. There was transmission throughout the State, with no particular seasonal pattern. Seroconversions detected in January, April to September and November to December. ***Incidence***. Incidence ranged from 60–90% in coastal areas to 20% in most inland areas except Cunnamulla, which had 90%. ***Disease***. Clinical disease commonly reported.
1999	***Transmission***. Normal pattern in most areas, but no activity on the northern Darling Downs (Dalby, Kingaroy). ***Incidence***. High incidence in northern and coastal areas (Clermont, 90%; Springsure, 95%; Maryborough, 75%;). ***Disease***. Reports of clinical cases from most areas. ***Features***. Prolonged wet summer and mild winter saw a high incidence in inland areas.

Western Australia

Normal features of bovine ephemeral fever virus transmission in Western Australia

BEF virus seroconversions will usually occur in sentinel cattle in the Kimberley region during the wet season (usually November–April). However, this appears to depend on the rainfall pattern and the general herd immunity within the Kimberley region, as some years have very limited BEF activity.

Some local rainfall–vector breeding patterns have been associated with late seroconversions (July to August) in sentinel cattle at Kununurra. BEF virus activity becomes more widespread in the southern part of the Kimberley region in epidemic years; if these correspond to high rainfall periods in the northern Pilbara area, some outbreaks of BEF can occur in cattle stations in this area.

Comments relevant to transmission in specific years are as follows.

BEF in Western Australia

Year	Description
1993	***Transmission***. None. ***Incidence***. None. ***Disease***. None reported or sent for laboratory diagnosis. ***Features***. Above-average rainfall in January and February; lower in March but a wet May. ***Comments***. The first bleed of sentinels had all seropositive for BEF, suggesting widespread infection in previous year(s) for this group.
1994	***Transmission***. At Kununurra, there were major seroconversions between November and January. At Kalumburu, there were seroconversions in November and a major seroconversion in April. ***Incidence***. At Kununurra the incidence of seroconversion was 14/15. At Kalumburu, the incidence was 3/11 in November 1993 and 7/7 to BT 1 in April 1994, but seroconversions still occurred in replacement cattle up to July 1994. ***Disease***. No reported disease. ***Features***. Rainfall in the region was well above average in December 1993 and February and March 1994. Local climate and geography effects at Kalumburu not directly associated with regional dry season. ***Comments***. Vector numbers did not reflect the heavy seroconversions at Kununurra this year. Perhaps the weather interfered with light traps.

Appendix 2

Outline of sampling procedure

This sampling procedure, established in 1969, describes the first use of sentinel herds for arbovirus monitoring in Australia (see Section 2.2). Sampling techniques have been slightly modified since that time.

Aim: To determine the epidemiology of known viruses in cattle in northern Australia.

Duration: Initially three years, then subject to review.

Cattle: Animals which are to be sampled are to be treated as far as practicable the same way as the rest of the herd and disposed of in time in the usual manner.

Number in group: Twenty from each successive calf crop born on the property, eg:

Year of birth	Number of calves	Total in group
1969	20	20
1970	20	40, less any deaths or sales
1971	20	60, less any deaths or sales

Times of sampling: The first bleed is to be made as soon as feasible after birth, then the calves are to be bled at intervals of not more than 3 months until first calving of heifers, then once a year. The exact spacing of the sampling times will depend on local conditions as it may not be possible to have the animals in hand at completely regular intervals. It is an advantage to have birth dates, either actual or approximate.

Identification of calves: If no reliable system of individual identification such as tagging, tattooing or fired numerals is already in use, 2 tags each with the same number to be put on at the time of the first sampling, one on each ear. Tags can be supplied by CSIRO if required.

Blood samples: A minimum of 1 oz of clean serum is necessary for immediate testing and long-term storage. The blood to provide this amount of serum could be obtained by means of a separate needle for each animal or by a clean tail bleed. Bottles and insulated transport boxes can be supplied by CSIRO if required. It is important that the serum is held at wet ice temperature or in a refrigerator until dispatch, then forwarded in an insulated container on ice to Long Pocket Laboratories, Brisbane, Air Express.

Tests: Tests will be made initially for antibodies to the following viruses: mucosal disease, infectious bovine rhinotracheitis, Myxovirus parainfluenza type 3, adenoviruses, and ephemeral fever virus. Other virus tests to be performed as they become possible or relevant.

18.12.69

Abbreviations and acronyms

BARC	Berrimah Agricultural Research Centre
BEF	bovine ephemeral fever
BLU	bluetongue (used to refer to virus serotypes)
BT	bluetongue (Office International des Epizooties code)
CPRS	Coastal Plains Research Station, 60km southeast of Darwin in the Northern Territory
CSIRO	Commonwealth Scientific and Industrial Research Organisation
DDRF	Douglas Daly Research Farm, 165km south of Darwin
EHDV	epizootic haemorrhagic disease virus
ELISA	enzyme-linked immunosorbent assay
EMAI	Elizabeth Macarthur Agricultural Institute
KRS	Katherine Research Station in the Northern Territory, 430km southwest of Darwin
NAHIS	National Animal Health Information Service
NAMP	National Arbovirus Monitoring Program
NCEEP	Northern Cattle Export Enhancement Project
NSW	New South Wales
NT	Northern Territory
PCR	polymerase chain reaction
PNG	Papua New Guinea
QIMR	Queensland Institute of Medical Research, Brisbane
Qld	Queensland
SA	South Australia
SO	southern oscillation
Tas	Tasmania
Vic	Victoria
VRRS	Victoria River Research Station, 450 km south of Darwin in the Northern Territory
WA	Western Australia

Glossary

'Adapted' virus Virus that has been adapted to a species other than the original species in which it was detected or to laboratory culture.

Adjuvant Adjuvants are oils, gels or other chemicals that cause a local cellular response in the tissue into which they are injected. When mixed with viruses they enhance the immune response. Sometimes, they leave a persistent lump.

Antibody A protein (immunoglobulin) that is formed in response to the presence of an antigen.

Antigen A substance that induces the formation of an antibody. In the diseases discussed in this book, the viruses act as antigens.

Arbovirus A virus that multiplies in arthropods and vertebrates in a cycle: vertebrate > arthropod > vertebrate. Arboviruses are usually able to multiply in a very limited range of species. They do not normally spread by contact in nature.

PCR Polymerase chain reaction. A highly sensitive test that can detect very small numbers of intact or fragmented virus particles.

Sentinel animal This is a previously uninfected, identified animal, kept at a specific location and used as a monitor of viral activity. Blood samples are collected at intervals to check whether a virus infection has occurred.

Seroconversion Appearance in the blood serum of antibodies following vaccination or natural exposure to an infected agent.

Serotype A subgroup of microorganisms identified by the antigens carried.

Vector A living organism (for the diseases discussed in this publication, an arthropod) that transmits an infectious agent from one host to another.

Viraemia The presence of viruses in the blood.

'Wild' virus This is virus that is circulating naturally and has not been modified by experimental manipulation. Less commonly called 'field virus'.

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