

Epidemiology of Bovine Ephemeral Fever in Australia 1981-1985

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Abstract

Bovine ephemeral fever is an important viral disease of cattle in Australia. The disease occurred each year, principally in summer and autumn, between 1981 and 1985. Queensland and the northern half of New South Wales were areas of greatest activity with only sporadic cases being reported from the Northern Territory and the northern third of Western Australia. Since 1981, the disease has been endemic in an extensive area of eastern Australia and has tended to occur in widely scattered outbreaks rather than the north-south advancing wave form of the epidemics of 1936-37, 1967-68, 1970-71 and 1972-74. The southernmost outbreaks between 1981 and 1985 were well within the limits of these earlier epidemics. The pattern of disease appears to have become seasonally endemic rather than periodically endemic in the northern two-thirds of eastern Australia. Ephemeral fever was not recorded in Victoria, Tasmania, South Australia or the southern part of Western Australia between 1981 and 1985.

The disease was most frequently reported in cattle under 3 years of age, but also occurred in older cattle.

Additional keywords: Kimberley virus; *Anopheles bancroftii*; *Culicoides brevitarsis*.

Introduction

Bovine ephemeral fever which is caused by an insect borne rhabdovirus, bovine ephemeral fever (BEF) virus, was first reported in Australia in 1936 (Mulhearn 1937; Seddon 1938). Reports cited by Mackerras *et al.* (1940) indicate that the disease was present before then. Since the 1936-37 epidemic, the disease has been characterized by major epidemics in 1955-56, 1967-68, 1970-71, 1972-74, 1974-76, separated by years with only small numbers of sporadic outbreaks (reviewed by St George 1981). More recently the epidemics have evolved from a disease pattern defined by Murphy (1974) as 'explosive epidemic' between 1967 and 1977 to 'slowly epidemic-appearing endemic' after that time. After 1977, the pattern was one of successive years of high and low prevalence of disease with epidemic occurrence each summer (Uren *et al.* 1983). Those cattle at risk were those that did not acquire infection in the previous epidemic. This paper describes the occurrence and epidemiology of ephemeral fever in Australia from 1 July 1981 to 30 June 1985.

Materials and Methods

Most of the information relating to the incidence of clinical ephemeral fever was summarized from the periodic reports of the Departments of Agriculture or Primary Industries of the respective States of Australia. The remaining information was provided by chief veterinary officers of the States or derived from personal observations. A consistent terminology in the localities from which outbreaks were reported was not possible as different systems were used in the field reports from State to State and even those from within a State. There is no form of standardized viral disease reporting within Australia. Absolute numbers of animals sick in a particular area are rarely recorded.

A questionnaire was distributed by a dairymans' organization in the Hunter Valley of New South Wales shortly after the termination of an outbreak of ephemeral fever in the local area in 1984. The initiative for the survey came from the organization although they asked for advice on what questions should be included. Each farmer was asked to record the number of cows affected by ephemeral fever by age, duration of clinical illness and mortality, and to estimate his production or financial loss.

In the winters of 1983, 1984 and 1985, a serological survey of cattle approximately 2–2½ years old was carried out in Queensland for another purpose. Their use for BEF serology was subsidiary. The sampling was based on the statistical districts used for data census (as listed in Table 1). The intention was to obtain serum samples for cattle born in each of the statistical districts in the winter months when ephemeral fever virus was not being transmitted. Many of the samples were collected at abattoirs.

Virus Isolation

Blood was collected from cows during fever and the leucocyte fraction was separated. Isolations of BEF virus from leucocytes were carried out either by the intracerebral inoculation of 1–2-day-old mice (St George *et al.* 1977) or by growth in Singh's *Aedes albopictus* cells for 1–2 weeks and then subculture in BHK-21 tissue culture (St George 1985). BEF viruses were identified by the microneutralization test using African green monkey kidney (Vero) tissue cultures (Cybinski *et al.* 1978) adapted by the use of antiserum prepared in rabbits against the BB7721 strain of BEF virus (Doherty *et al.* 1969).

Serology

Microneutralization tests for antibodies on survey sera were carried out by the method of Cybinski *et al.* (1978) with the substitution of the BB7721 strain of BEF virus or the CSIRO 368 strain of Kimberley virus (Cybinski and Zakrzewski 1983). A dilution of 1 : 4 was used for all the survey sera. Seroconversion describes the change in neutralizing antibodies to ephemeral fever virus from negative to positive in consecutive serum samples when measured at a dilution of 1 : 4, positive samples then being titrated. The first serum sample was collected during fever and the second 1–2 weeks later.

Results

The results of the test on sera collected for surveys in Queensland are shown in Tables 1 and 2. Forty-one questionnaires were received from dairy farmers in the Hunter Valley and the results are summarized in Table 3. The farmers reported that the whole herd production fell by 5–20% for 4 weeks while ephemeral fever was present in the herd. They would not disclose production figures.

Reports of clinical disease are grouped within yearly intervals, from 1 July in one year to 30 June in the next. There were no cases of clinical ephemeral fever in Tasmania, Victoria, South Australia or the southern two-thirds of Western Australia. Negative serological tests in excess of 20 000 per year for certification of live cattle exports, provided confirmatory evidence BEF virus had not occurred in the southern third of the Australian continent. In the few instances where antibodies were found in the sera of cattle from the southern part of the continent, the animals had been imported from another State where outbreaks had been recorded. There was no evidence of secondary local spread.

Table 1. Annual prevalence of bovine ephemeral fever virus neutralizing antibodies in 2-3-year-old cattle in Queensland tested between 1983 and 1985

Area	1983	1984	1985
Moreton	15% (3/20)	20% (4/20)	10% (2/20)
Darling Downs	15% (3/20)	26% (6/23)	5% (1/20)
Wide Bay-Burnett	5% (1/20)	45% (9/20)	6% (1/18)
South-west	0 (0/20)	0 (0/16)	10% (1/10)
Fitzroy	5% (1/20)	10% (2/20)	48% (12/25)
Central-west	15% (3/20)	16% (4/24)	7% (1/15)
Northern	20% (4/20)	30% (6/20)	47% (9/19)
North-west	15% (23/149)	14% (16/144)	15% (27/155)
Far north	8% (2/25)	5% (1/19)	—
Mackay	60% (12/20)	55% (11/20)	41% (9/22)
Total	16% (52/334)	18% (59/326)	21% (63/304)

Table 2. A comparison of the results of virus neutralization tests for antibodies to ephemeral fever and Kimberley viruses for survey cattle serum diluted 1:4

Year	KIM+ BEF ^{-A}	KIM- BEF ^{+B}	KIM+ BEF ^{+C}	KIM- BEF ^{-D}
1983	60	18	34	222
1984	71	10	49	162
1985	39	22	41	202
	170 (18%)	50 (5%)	124 (13%)	586 (63%)

^A Serum positive to Kimberley virus, negative to BEF virus.

^B Serum positive to BEF virus, negative to Kimberley virus.

^C Serum positive to Kimberley virus, positive to BEF virus.

^D Serum negative to Kimberley virus, negative to BEF virus.

Table 3. Summary of the results of a Questionnaire on the effects of an epidemic of bovine ephemeral fever in the Hunter Valley of New South Wales in the summer of 1983-84

Age	No. of cows observed	No. died	No. sick > 3 days	No. sick ≤ 2 days	No. unaffected	% sick or died
Fourth calf	391	8	34	79	270	31
Third calf	178	4	29	101	44	75
Second calf	178	1	45	115	17	90
First calf	256	4	45	136	71	72
12 months	171	2	2	71	96	44
< 12 months	183	0	5	33	145	21
Total	1357	19 (1.4%)	160 ^A (11.7%)	535 ^A (39.4%)	643 (47%)	

^A 5.1% of the sick cows aborted

1981-1982

The first confirmed report of the disease following the winter months of 1981 came from south-east Queensland during September. This was followed by reports of sporadic cases of ephemeral fever in south-east Queensland during

November with the numbers of reported cases increased markedly towards the end of December. During this time isolated mortalities occurred and it was reported that the outbreak was characterized more by lameness and recumbency than by other signs. As in previous years, the highest incidence was in cattle in the 1½–2½ year age group.

Heavy and widespread falls of rain during January 1982 coincided with many reports of outbreaks of ephemeral fever. The locations referred to in the text are shaded in Fig. 1. Again, it was reported that cattle of the 12–24 month age group were chiefly affected with a case fatality rate of 1%. It was observed that the cattle appeared to be more severely affected clinically than those involved in previous outbreaks.

Clinically affected animals were observed in New South Wales from late February through to early June (Fig. 1*a*). However, more cases were observed in March and April mainly in heifers and young bulls. Isolated incidents occurred at Bourke (Western Plains) and West Wyalong (Central Slope Division) following extensive flooding which followed the prolonged drought.

Confirmatory tests. Between 2 and 18 January 1982, BEF virus was isolated from cattle with typical ephemeral fever in three herds separated by 100 km in south-east Queensland. Seroconversions occurred at Peachester in 11 out of 124 cattle aged 7–22 months which converted between 26 December 1981 and 29 January 1982. In New South Wales, serological confirmation was obtained for clinical cases in each location where the disease was observed, including the cases in inland areas.

1982–1983

No reports of ephemeral fever were received until October when two outbreaks were diagnosed clinically in the Maryborough Division. Drought conditions developed throughout the State. There were no further reports of the disease until January 1983 when a severe outbreak occurred on the Atherton Tableland of north Queensland involving cattle from 1 to 3 years of age on most properties. Approximately 20% of dairy cattle and 10–15% of beef cattle were affected. Occasional deaths in bullocks were reported. Isolated cases were diagnosed in south-east Queensland. During the period April to June 1983 ephemeral fever was clinically diagnosed as shown in Fig. 1*b*.

In New South Wales, a resurgence of ephemeral fever occurred in the Hunter Valley (Central Coastal) at the end of the winter. Sporadic cases were initially observed at Taree and Gloucester in July. During August there was a localized epidemic in the Dungog area where each of a number of dairy farmers reported that up to 50% of his herd was affected. Clinical signs were relatively mild with lameness and agalactia the most noteworthy findings. There were no further reports on ephemeral fever until February 1983. Sporadic cases were then reported on various areas of coastal New South Wales until the onset of winter.

Confirmatory tests. No BEF isolations were attempted in this period. However, in a sentinel herd at Kairi, north Queensland, 24 out of 42 cattle aged 1–3 years seroconverted between 10 January 1983 and 16 June 1983. In New South Wales, serological confirmation was obtained for the winter–spring outbreak in the Dungog area and for the cases on the south coast in March.

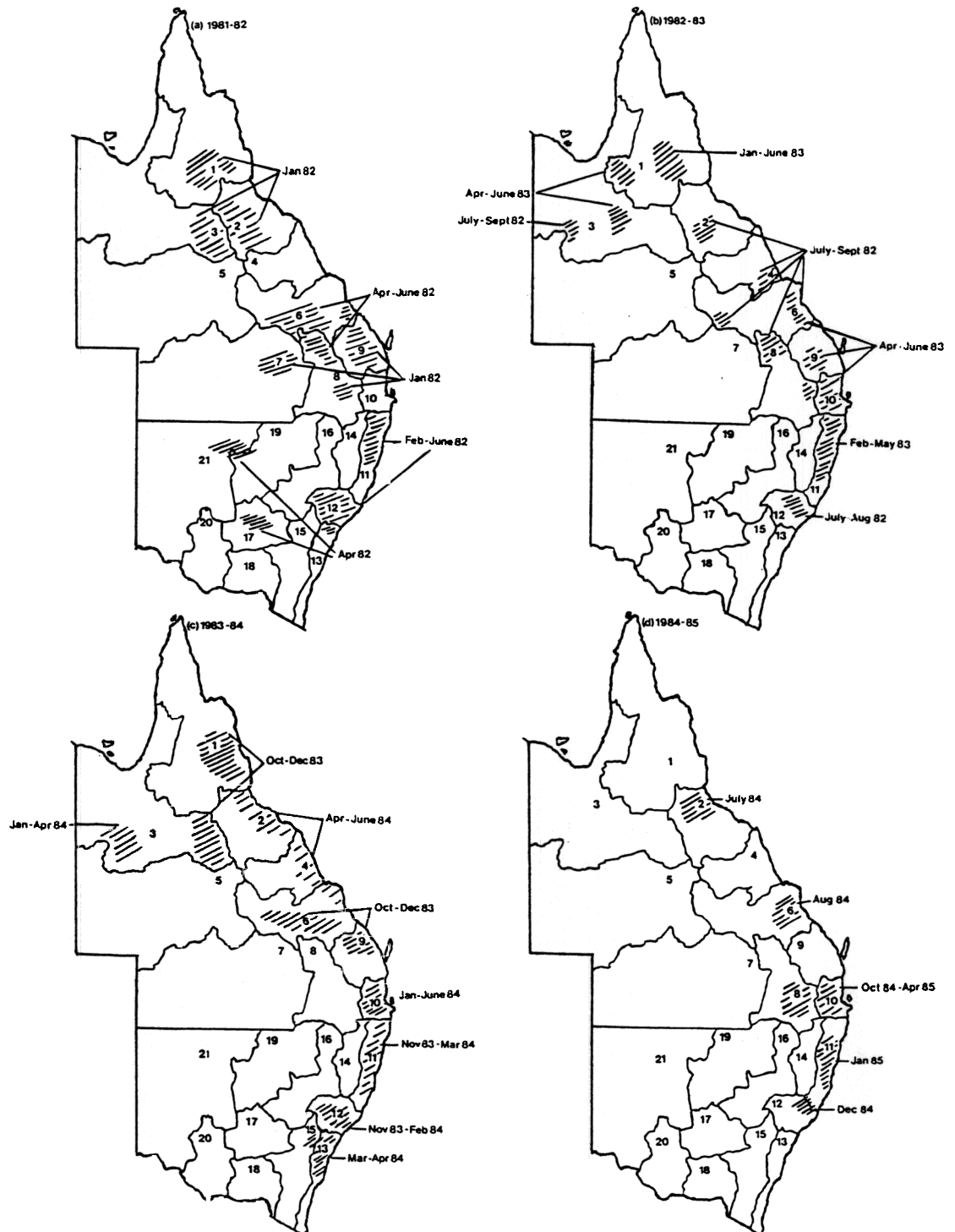


Fig. 1. Areas in which ephemeral fever was reported during the years (a) 1981-82, (b) 1982-83, (c) 1983-84, (d) 1984-85 are hatched. The areas correspond to the names of the towns or localities which appear in the text. The statistical agricultural divisions of Queensland and New South Wales are numbered as follows: (1) Far North, (2) Northern, (3) North West, (4) Mackay, (5) Central West, (6) Fitzroy, (7) South West, (8) Darling Downs, (9) Wide Bay-Burnett, (10) Moreton, (11) Northern Coastal, (12) Central Coastal, (13) Sydney and Southern Coastal, (14) Northern Tableland, (15) Central and Southern Tableland, (16) Northern Slope, (17) Central Slope, (18) Southern Slope, (19) Northern Plains, (20) Southern Plains, (21) Western Plains.

1983-1984

The wet conditions which prevailed during the July-September quarter in Queensland coincided with an increased prevalence of ephemeral fever when the disease would normally have been expected to be quiescent. Outbreaks confirmed by seroconversion of paired sera were reported at Charters Towers, Kairi, Chinchilla, Stuart, Mt Isa, Alpha, Marlborough and St Lawrence. It was reported that cattle under 3 years of age were chiefly involved. With good seasonal conditions prevailing during October-December the disease was diagnosed clinically in coastal areas and adjacent hinterland from the Atherton Tableland to the Queensland-New South Wales border. In central Queensland a widespread epidemic occurred which commenced at Clermont in late September and extended to Rockhampton and coastal areas in early November.

Following generalized rains in early January, the disease was diagnosed in herds located on the east coast and adjacent hinterland, from the Atherton Tableland to the Queensland-New South Wales border. After further generalized rain, the incidence of the disease increased markedly in south-east Queensland. During the first quarter of 1984, prevalence rates ranged from 5 to 25% with a mortality rate on a whole herd basis of 1%. During April to June outbreaks of ephemeral fever were diagnosed at properties in central and southern Queensland.

In New South Wales ephemeral fever was first observed at Paterson in the Hunter Valley and at Casino, areas 350 km apart. A severe epidemic spread through the Gloucester and Taree areas near the coast during December, the lower Hunter Valley during January-February, and in the upper Hunter Valley as far as Muswellbrook in February-March. The disease affected cattle in the dairying areas west and south of Sydney during March and April which spread as far south as Milton. Relatively few cases were reported on the north coast of New South Wales during this major epidemic.

A particular feature was the severity of the disease as illustrated in Table 3. The loss of production of the whole herd assessed by the owners in the questionnaires varied from 5 to 50%. In most cases absolute loss was not given. The period over which this loss occurred varied from 2 to 6 weeks depending on the intensity of the outbreak. Most of the deaths were in cows with high milk yield. The 36 cows which aborted did so in late pregnancy (8-9 months) and failed to come into lactation on recovery, thus losing 1 year's production of milk.

Confirmatory tests. Three strains of BEF virus were isolated from cattle with ephemeral fever in one herd in south-east Queensland between 21 and 25 January 1984 and serology showed that 23 of the 40 cattle without anti-bodies to BEF virus aged 6-42 months, seroconverted between 16 December 1983 and 7 February 1984. Serological confirmation was obtained in New South Wales for approximately 70 cases scattered throughout the epidemic area between 1 November and mid-April (Fig. 1c).

1984-1985

Sporadic outbreaks of ephemeral fever were reported in the Townsville area in July, then in the basin of the Fitzroy River in the central east coastal region in August. The outbreaks in Queensland followed a few weeks after local heavy rain, which is a very unusual occurrence in these areas during winter. In the summer, between October 1984 and April 1985, small outbreaks were reported in south-east

Queensland. In December, cases occurred on the eastern Darling Downs but a major epidemic did not develop. It was observed that clinical cases were more severe than in recent earlier epidemics. In the sentinel herd at Peachester 10 out of 30 susceptible cattle (those without antibody to BEF virus and no history of disease) aged 1–3 years seroconverted to BEF virus between 17 January 1985 and 18 April 1985. However, only three clinical cases were noted. Confirmed, sporadic cases were reported from Taree and Grafton of northern coastal New South Wales in December 1984 and January 1985 respectively (Fig. 1*d*).

Western Australia and Northern Territory

Since June 1981, there have been few reports of clinical cases of ephemeral fever in Western Australia. Sporadic cases were reported in the Kimberley region in March 1985. Larger outbreaks involved 2½–3½-year-old cattle in a herd of 1100 from which a 33% overall morbidity was reported and almost the whole of a herd of 400 similar cattle in April, approximately 1000 km south-west of the first location, at Wallal which is near the west coast. All were clinical reports, unconfirmed by serology or virus isolation. Similarly, in the Northern Territory, 1983–84 was the first year since 1980 that clinical ephemeral fever could be serologically confirmed although the number of cattle involved is unknown. However, it is now considered from sentinel herd observations (G. P. Gard, personal communication), that BEF virus is active each year and that most Northern Territory cattle are infected before maturity, but do not show clinical disease. In the Katherine and Darwin areas clinical disease regarded as ephemeral fever is observed every wet season (between January and March).

Discussion

The study of the epidemiology of any disease is facilitated by standardized procedures preferably with a prospective plan for assembling hard data. Carried out on a national basis, there is then a firm base for sophisticated analysis, which will reveal even small changes of prevalence or age, geographical and seasonal distribution. There is no standardized system of reporting ephemeral fever, or other virus diseases of cattle in Australia. In the absence of such a system, the problem remains to collect what information has been recorded before memory of its existence or location fades and to add the direct observation of the writers. At best, it is opportunistic epidemiology but it does give a broad picture of the disease from which limited conclusions may be drawn.

From the point of view of the veterinarian or field officer of a Department of Primary Industries who observes clinical ephemeral fever, confirmation of his diagnosis (3–4 weeks later) contributes nothing to his problem. The advice to the owner, and treatment of the affected cattle, have to be provided immediately. However, most of the observers whose reports have been examined are familiar with ephemeral fever because of its clinical effect on herds and because it has been seen very frequently in most affected districts since 1968.

The characteristic clinical history is the basis for reports of ephemeral fever. However, examination of sera from individual cows in two sentinel herds in north and south Queensland indicated that not every animal with a febrile illness at the time of an epidemic of ephemeral fever could be confirmed as suffering from this disease. For instance, BEF virus was not isolated nor did seroconversion for BEF

antibodies occur in 5 of 20 cows which had fever and an illness which was classed as ephemeral fever by the owner, a veterinarian, and the person who collected the blood samples (T. D. St George, unpublished data). It is also possible that not all the cows with ephemeral fever develop an adequate antibody response. Two instances were reported by St George *et al.* (1977) where BEF viruses were isolated from cows with typical ephemeral fever but which failed to seroconvert to the strain of BEF virus used in the neutralization test. A more intensive study would have to be mounted to determine whether additional viruses can produce clinical signs resembling those of ephemeral fever.

In each of the years since the previous report, which covered the period from 1977 to 1981 (Uren *et al.* 1983), clinical disease occurred in eastern Australia with a particularly severe epidemic in the Hunter Valley of New South Wales in 1983–84. The occurrence of disease in sentinel herds was confirmed by the isolation of BEF virus and serology in south-east Queensland in the summer of 1981–82, by serology in North Queensland in 1982–83, by virus isolation and serology in south-east Queensland in 1983–84, and by serology in south-east Queensland in 1984–85. Serological confirmation has been provided for clinical cases in New South Wales in each of the 4 years under study.

A north–south time sequence of outbreaks continued in the summer of 1981–1982. However, the outbreaks were scattered, unlike the form of epidemics in the early part of the 1970–1980 decade where cases occurred over a broad band of country almost simultaneously. The localized epidemic in the Hunter River valley of New South Wales appeared to develop independently of the general north–south moving epidemic as it has done since 1972 (St George *et al.* 1977; Kirkland 1982; Uren *et al.* 1983).

The maps which illustrate the epidemics of 1936–37 by (Seddon 1938; Murray 1970; St George *et al.* 1977), and to a lesser extent by Uren *et al.* (1983), show a sequence of times in which cases were reported which indicated a general north–south direction of movement. This clear pattern of movement was not apparent between 1981 and 1985 a period marked by lowered summer rainfall and frequent droughts, in contrast to the generally high summer rainfall of the early 1970s.

The severity of the disease in dairy areas is known from anecdotal reports but is shown more clearly by the data provided by dairymen in Table 3. The overall mortality rate of 1.4% was limited to the highest producing cows and heavy bulls. In all, 57% of cows became recumbent, but did not die. Average herd production loss of 20% would include the production from the unaffected cows. The loss in production of those cows with ephemeral fever would therefore be higher than 20%. Davis *et al.* (1984) found that the average production of 15 individually metered cows in a herd fell by 70% during acute illness compared with the level 1 week after recovery. The loss of milk for the balance of lactation in the Hunter Valley herd would be concealed by the pooling of production of affected and unaffected cows. The abortion rate of 5.1% is high and merits investigation to determine the pathogenesis. From the comments of the owners, abortions tended to be in the eighth or ninth month, and the affected cows did not come into useful lactation, which meant a complete loss of one year's production as well as the calf. Parsonson and Snowdon (1974) provided experimental data that BEF virus does not cross the placenta, so the abortion may be induced indirectly rather than the virus killing the fetus. There was no suggestion that the abortions were due to

concurrent Akabane virus infection. In 1984 there was a high level of immunity in the cattle population following the epidemic of the previous year (Kirkland and Barry 1984). In addition, the congenital abnormalities characteristic of Akabane virus infection were not described in any of the fetuses although these abnormalities are very familiar to both farmers and veterinarians in the Hunter Valley of New South Wales.

The serological surveys of Queensland cattle (Table 1) are small, but indicate that BEF virus infection has occurred over much of the State during a time span of 3 years. Antibody to the virus was not detected in the far south-west of the State, an area of very low (<250 mm per year) rainfall. In comparison, the Mackay statistical division, an area with a very high annual summer rainfall (approximately 1500 mm per year) had the highest prevalence in the three successive years. The results of the serological survey (Table 1) also need further comment in terms of comparative tests on the same sera in Table 2, which were subjected to comparative tests with Kimberley virus (St George *et al.* 1984). Cybinski (1987) has reported that infection with Kimberley virus causes the production of homologous antibodies to Kimberley virus and may cause the production of low level antibodies to BEF virus in some cattle, but infection with BEF virus induces the formation of antibodies to both BEF and Kimberley viruses. St George *et al.* (1984) illustrated that a higher titre to Kimberley virus could exist in a cow which has had a recent infection with BEF virus. Thus the 13% of sera (Table 2) which were positive for both Kimberley and BEF antibodies may include an unknown proportion which do not reflect exposure to BEF virus. All these sera will be examined by a series of further tests to determine which virus infections were responsible for generating the antibodies. In spite of this reservation, an overall summation of the results of the survey is that most animals of approximately 2-2½ years of age when sampled in Queensland in 1983, 1984 and 1985 had escaped infection with BEF virus. This suggests that there is incomplete immunity in particular age groups, providing a source of susceptible animals for infection later in life when they respond more severely.

The low prevalence of antibodies to BEF virus in younger age groups revealed by the serological survey seems to be borne out by the age prevalence of clinical cases in the severe epidemic in the Maitland district of New South Wales in 1984 (Table 3). However, there were 31% of 391 cows which were 6 or more years old with clinical disease (Table 3). The cases in the older animals could be the result of individual cows not having encountered disease in earlier epidemics in the district (St George *et al.* 1977; Kirkland 1982; Uren *et al.* 1983), or perhaps due to inter-current infection with other arboviruses as suggested by St George (1985), or a waning of immunity with age.

An association with recent rain is reported by some observers of outbreaks of ephemeral fever, but not others. In the winter of 1985, the general association was strong in coastal Queensland. In most of the strip of country between the north-south Great Dividing mountain ranges and the sea the normal yearly rainfall occurs almost entirely in the summer months. Heavy winter rains are a rarity. The day-time temperatures are high although night-time frosts can occur in June and July in a severe winter. The unseasonal rains recorded near Townsville and Rockhampton were closely followed (3-4 weeks later) by epidemics in the regions around those centres. Association with recent heavy rain has been reported by various authors

(Seddon 1938 and Murray 1970). The epidemics of 1955 and 1956 (Albiston 1966) occurred in years of exceptionally heavy rains over the whole of eastern Australia. In Kenya, Davies *et al.* (1975) and Davies (personal communication) are convinced of an association with recent rain and the incidence of BEF. However, Murray (1970) and Davies *et al.* (1975) state that the disease did occur in drought areas or areas where recent rain has not fallen, perhaps as a spill-over from areas experiencing rainfall. Murray (1970) showed that flooded rivers flowing through dry areas can be a local factor in the spread of the disease. However, the general wind flow patterns from epidemic areas determined the duration of distant movements into such areas in inland Australia.

BEF virus was isolated from *Anopheles bancroftii* (Standfast and Muller 1984) and *Culicoides brevitarsis* (H. A. Standfast, personal communication) collected at Peachester, south-east Queensland, during February 1984. These findings provide possible vector species for coastal Queensland and New South Wales, but not the inland where these species do not occur or are in low density. A mosquito vector is more directly affected by recent rainfall than *C. brevitarsis*, but the reports of cases are not good enough to determine the time lag between rainfall occurrence and the time of the first case.

Both farmers and disease control officers tend to regard ephemeral fever as a familiar problem that cannot be prevented, so is not worth reporting and data is lost. The sentinel herd network (St George *et al.* 1977; St George 1980; Uren *et al.* 1983) does not exist as an independent and continuous reporting system to provide clinical reports, and serological confirmation. The apparent change in the pattern of ephemeral fever which occurred in the period covered by this paper demonstrates clearly that data on a familiar disease should be recorded and reported as a matter of routine. However, even in relatively quiet years a prevalence of 16–21% of many millions of cattle in the area of its occurrence with a disease which can be either very mild, severely disabling or fatal, can decrease the overall efficiency of the national production of milk and meat.

In spite of the inadequacies of the existing reporting systems, there are several conclusions which can be drawn from the information presented here. Ephemeral fever has occurred in each of the years from 1981 to 1985; where specimens have been submitted to a laboratory it is generally confirmed by serology and occasionally by isolation of BEF virus; the neutralization test may detect false positives in serological surveys; it is substantially a summer disease; winter outbreaks in central and northern Queensland followed heavy unseasonal rain; the disease has occurred well north of the southern limits of earlier epidemics; and that many cattle are escaping infection each year in the overall area where the disease has occurred, thus maintaining a reservoir of susceptible cattle. A large area of eastern Australia should now be regarded as endemic for ephemeral fever. Consequently prospective steps should be taken to obtain objective data in this area on the incidence and economic effect in representative herds of both beef and dairy cattle.

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