Bluetongue and Douglas virus activity in New South Wales in 1989: further evidence for long-distance dispersal of the biting midge Culicoides brevitarsis

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SUMMARY: Infection of cattle with bluetongue and Douglas viruses was detected on the central and southern coast of New South Wales from January to April 1989. Bluetongue virus infection was found well south of areas of expected occurrence. Evidence is presented to support wind-borne dispersal of infected vectors, Culicoides brevitarsis, southwards from the Hunter Valley.

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Introduction

Murray (1987) described a method whereby epizootics caused by oroviruses transmitted by the biting-midge Culicoides brevitarsis could be analysed in retrospect. An analysis of the Akabane disease outbreak in New South Wales in 1983 showed the likelihood of wind dispersal of infected vectors inland from the Hunter Valley. We now report an analysis of the activity of 2 viruses, bluetongue and Douglas viruses, in coastal southern New South Wales in 1989.

Materials and Methods

Sentinel herds, comprising antibody-free young cattle, are established annually in New South Wales. In the 1989 season, 27 such groups of 5–7 month old cattle were distributed to cover areas where C. brevitarsis is endemic or absent, with a higher proportion within or just outside its southern distribution. This paper deals with the results of this analysis. We used a vector ESF from the central coast of New South Wales, which is abundant in Bega on the far south coast where it is usually absent (Murray 1987). Blood samples were taken monthly from these cattle and tested for antibodies to a variety of viruses (Kirkland et al. 1992). In 1989, bluetongue (BTV) and Douglas viruses were transmitted with high frequency. Cattle were considered to have been infected with BTV if they gave a positive result in the competitive enzyme-linked immunosorbent assay (ELISA) (Jeggio et al. 1992) and with Douglas virus if their neutralising antibody titre was greater than 16.

The areas of New South Wales potentially favourable for multiplication of C. brevitarsis were determined and the degree of favourableness expressed in a scale of 1 to 6 (as described by Murray and Neis 1987). A 'brevitarsis-line' was calculated for each of the 2 viruses. These lines depict a probable southern limit of overwintering populations of this vector that could develop into populations capable of sustaining activity of the particular virus. C. brevitarsis is a vector of a number of viruses belonging to the bluetongue serogroup of the orbiviruses and the Simbu serogroup of the genus Bunyavirus; these include BTV and epizootic haemorrhagic disease of deer (EHD) of the former and Akabane and Douglas of the latter (Muller et al. 1982).

It is, however, in experimental studies a poor vector of EHDV (Muller 1987) and in New South Wales viral activity is usually restricted to the northern rivers region of the coastal plains. The values used in the calculation of the 'brevitarsis-line-bluetongue' were selected to reflect this distribution, an approach first described by Murray (1982).

Results

The area of interest was the central and southern coast of New South Wales, within which there were sentinel herds at Taree and Gloucester in the Manning River basin, Paterson, Singleton and Scone in the Hunter Valley, Richmond and Camden, and Milton, Bodalla and Bega on the southern coastal plains.

Douglas virus (Figure 1)

All cattle infected at Taree, Gloucester and Paterson by the end of January, half the herd at Camden during February and April and all as far south as Milton by the end of March. The 'brevitarsis-line-Douglas' extended as far south as Nowra to the immediate north of Milton, and the final distribution of virus reflected the favourable conditions that developed within the defined region during the summer.

Bluetongue Virus (Figure 1)

About 4% of the sentinel herd at Taree seroconverted during January, 90% and 20% at Taree and Gloucester, respectively, at the end of February, and all by the end of March when 10% of those at Paterson in the Hunter Valley had also seroconverted. During April half the cattle at Paterson, Camden and Milton seroconverted. The 'brevitarsis-line-bluetongue' extended only to Taree and although conditions approached being favourable in the Hunter Valley, they remained unfavourable further south. Clearly, there was a sudden extension of BTV activity into an unsuitable area. The rapidity of the extension and the extent of the region involved made movements of infected stock an improbable cause. Dispersal of vectors by wind appeared likely.

Weather Patterns

The weather patterns from January to April 1989 were appraised. In early March there were 8 consecutive evenings and nights that were warm and humid with steady winds from the north-north-east along the coastal plains. These winds were associated with a stationary high pressure cell over the Tasman Sea. This pattern was repeated later in the month on 2 evenings.

Discussion

Infections with Douglas virus followed a pattern that reflected the probable distribution of C. brevitarsis in adequate numbers for transmission of the virus. The 'brevitarsis-line' for Douglas virus is contrast, BTV infections were suddenly found in southern areas where C. brevitarsis was unlikely to be present in adequate numbers. Analysis of the 1983 epidemic of Akabane disease showed that infected vectors were probably dispersed by wind into areas 150 to

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200 km from a major source of infection in the Hunter Valley (Murray 1987). The weather systems that occurred in March 1989 could have blown infected vectors south and along the coastal plains from the Manning River basin, where there had been extensive transmission of BTV. This region is just north of the Hunter Valley, where there had also been much transmission of BTV. Of particular interest is that the data cover 2 viruses with the same vector. Infections with one developed as conditions became favourable for the vector/virus cycle, and superimposed upon this distribution was that of another virus later in the season, the apparent result of a sudden invasion from the north. The wind systems responsible could have also spread Douglas virus further south. Thus, it appears that invasion of vectors infected with viruses from the Hunter Valley to the southern coastal plains is possible. Such movements complicate epidemiological interpretations of disease outbreaks or viral activity. These need to be appraised critically on each occasion.

In the past there have been outbreaks of Akabane disease in the Camden area and further south (Hartley et al 1977; Haughey et al 1988). It is possible that some of these outbreaks may have resulted from wind dispersal of infected vectors.

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References

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