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Trajectory analysis of winds and vesicular stomatitis in North America, 1982–5

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SUMMARY

Outbreaks of vesicular stomatitis, serotype New Jersey, during epidemics in the United States and northern Mexico, 1982–5, were examined by backward trajectories of winds to investigate spread and possible sources. The outbreaks selected for analysis did not involve introduction of disease by infected animals. The findings indicate that wind could have been responsible for carrying infection from northern Mexico to Arizona and New Mexico and thence to Colorado and Utah and on to Wyoming, Idaho and Montana. The results of these analyses are consistent with the findings from T1 RNAse fingerprinting of virus isolates from outbreaks during the epidemics. The arrival of the trajectories was associated with the passage of a front and rain or passage of a front alone or rain alone. At the time of the trajectories temperatures of 10 °C and higher were recorded at heights up to 2500–3500 m.

Introduction by airborne particles would appear unlikely as it would have required a source of at least 10^5 infectious units per minute per animal. Vesicular stomatitis virus had been isolated from *Simulium* and *Culicoides* during the epidemic with amounts of virus from *Simulium* sufficient to suggest biological transmission. The possibility of *Simulium* infected with vesicular stomatitis virus being carried downwind to introduce disease is discussed in relation to the behaviour of *Simulium* and the pathogenesis of vesicular stomatitis in large animals.

INTRODUCTION

In 1982–3 there was a major epidemic of vesicular stomatitis (VS) in the western United States and further outbreaks occurred in 1984, 1985, and 1986 ([1, 2]; M. A. Mixson, personal communication, 1988). The serotype responsible was New Jersey. There had been previous outbreaks of VS, New Jersey serotype, in some of the western states in 1966 and 1972 [3, 4]. Outbreaks of VS, New Jersey serotype, were reported in the northern Mexican states of Sonora (1982, 1984 and 1985) and Chihuahua (1981, 1982, 1984 and 1985) ([5]; J. Mason, personal communication, 1989).

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The origins, methods of spread and persistence of vesicular stomatitis virus are controversial; spread has been ascribed to movements of animals, biting and non-biting flies, aphids or leafhoppers and persistence to persistent infection of vertebrate hosts or the presence of virus on pastures [6–9].

A number of investigations were carried out during and after the 1982–6 outbreaks. Nichol [10–12] carried out T1 RNase fingerprint analyses of isolates from USA and Mexico between 1982 and 1986 and found that within the USA epidemic outbreaks (1982–3, 1984 and 1985–6) the virus populations were genetically relatively homogeneous and that Mexican viruses isolated in the same year as the US epidemics had similar RNA fingerprints. From these results he concluded that Mexico could have been the origin of the US epidemics. Field investigations showed that disease spread through movements of animals and that in herds infection and lesions could continue for up to 2 months [1, 13]. However there was spread of disease over long distances without intermediate outbreaks [1, 14]. During the 1982 outbreaks there were reports of swarms of *Simulium*; VS virus, New Jersey serotype, was isolated from the biting flies, *Simulium* and *Culicoides*, in Colorado and it was suggested that these insects could be involved in transmission of VS virus [1, 8, 14–16].

In a previous investigation Sellers and Maarouf [17] found that possible sources and spread of western equine encephalitis in USA and Canada could be traced by analysis of backward trajectories of winds. It was therefore decided to analyse the trajectories of winds at the time of the vesicular stomatitis (New Jersey) outbreaks in western USA and northern Mexico. The aim was to find out if outbreaks, whose origin could not be explained by movement of infected animals or continual infection, could have resulted from the introduction of VS infection on the wind.

BACKGROUND DATA

Horses, cattle and wild ruminants are the main species affected with vesicular stomatitis in the western United States. In cattle inoculation of the tongue with VS virus results in lesions in 1–3 days; on farms or in experimental in-contact infection the disease interval in horses and cattle may vary from 3–15 days [6, 13, 18]. After the initial infection disease may persist on a farm for up to 66 days [1, 13]. Virus has been isolated 3 weeks after the initial lesion and shedding of virus from an infected animal may occur 38 days after the last known observation of lesions [13]. Despite the presence of antibody, reinfection of animals with the same virus serotype can occur 30–60 days after recovery from VS [6].

The backward trajectories of winds were computed every 6 h up to 120 h (5 days) starting at three levels: 1000, 900 and 850 mb at approximate heights of 0·1, 1·0 and 1·5 km above sea level respectively. A three-dimensional trajectory model [19] was applied as described previously [17]. Trajectory analysis was carried out on the first outbreaks in particular areas. A period of 3–15 days before the date of the outbreak was examined to take account of variation in the disease intervals and delays in reporting. In most instances backward trajectories were started at 18.00Z (18.00 Greenwich Mean Time (GMT), i.e. 11.00 Mountain Standard Time (MST)).

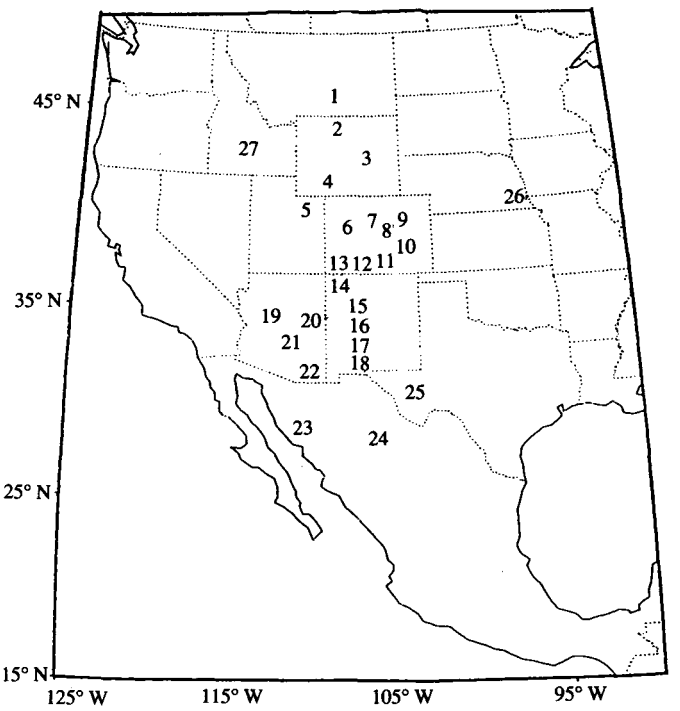

Fig. 1. Location map of North America showing VS outbreaks (1982–5) selected for trajectory analysis or as sources. 1982. Arizona; 19, Camp Verde; 21, Mesa. Colorado; 9, Adams Co., 11, Alamosa Co.; 12, Archuleta Co.; 8, Denver; 13, Durango; 7, Jefferson Co.; 6, Mesa Co. Idaho; 27, Jerome Co. Montana; 1, Yellowstone Co. New Mexico: 15, Albuquerque. Utah: 5, Uinta Co. Wyoming: 2, Bighorn Co.; 3, Converse Co.; 4, Sweetwater Co. Mexico: 24, Chihuahua; 23, Sonora. 1983. Nebraska: 26, Otoe Co. 1984. Texas: 25, Marfa. 1985. Arizona: 22, Fort Huachuca; 20, Pinetop. Colorado: 10, Pueblo. New Mexico: 14, Aztec; 18, Los Cruces; 16, Los Lunas; 17, Truth and Consequences. Mexico: 24, Chihuahua; 23, Sonora.

ANALYSES

VS in 1982

The first outbreak of VS, New Jersey, was recorded on 23 May 1982 at Camp Verde, Arizona. Subsequently disease occurred in June at Albuquerque, New Mexico and in July in north-western New Mexico, eastern Utah and western Colorado. In August VS was found across the Continental Divide of the Rocky Mountains in eastern Colorado and Wyoming; in late August it was in Idaho and in September in Montana ([1]; G. A. Erickson, personal communication, 1988). Such spread could not be accounted for by animal movements alone. VS was reported in April 1982 in Sonora state, Mexico, and in June 1982 in Chihuahua state, Mexico ([5]; L. C. Vanderwagen, personal communication, 1986; J. Mason, personal communication, 1989). The location of some of these outbreaks are shown in Fig. 1.

The first outbreaks in areas of the above states were subjected to trajectory analysis. In Table 1 the possible sources of the wind as calculated by backward

Table 1. *Source of backward trajectories at 850, 900 and 1000 mb up to 30 h previously together with weather conditions on arrival for outbreaks of vesicular stomatitis in western USA and in northern Mexico in 1982–3*

Location and date of disease	Date of arrival	Source at 850, 900 and 1000 mb	Interval (days)	Conditions
Camp Verde AZ* 23 May 1982 Fig. 2*a*	18 May 1982 3B§	18† NW Sonora Mexico	5	TF‡
Chihuahua, Mexico 4 June 1982	30 May 1982 3C	24 W Sonora Mexico	5	TF
Albuquerque, NM 22 June 1982 Fig. 2*b*	14 June 1982 3D	24 NW Sonora Mexico	8	TFR‡
Mesa, AZ 10 July 1982	5 July 1982	12 SW Arizona	5	TFR
Mesa Co., CO 21 July 1982 Fig. 2*c*	16 July 1982 3E	13 NW Arizona	5	TFR
Durango, CO 22 July 1982	16 July 1982 3F	12 C Arizona	6	TFR
Uinta Co., UT 28 July 1982	23 July 1982 3G	24 N Arizona	5	TFR
Archuleta Alamosa Cos., CO 1–4 Aug. 1982	27 July 1982 3H	18 N New Mexico	5–8	TFR
Jefferson Adams Cos., CO 11 Aug. 1982 Fig. 2*d*	3 Aug. 1982 3J	12 W Colorado	8	TFR
Sweetwater Co., WY 11 Aug. 1982	3 Aug. 1982 3K	9 NE Utah	8	TFR
Converse Co., WY 12 Aug. 1982	2 Aug 1982 3M	12 NE Utah‖	10	TFR
Bighorn Co., WY 17 Aug. 1982	11 Aug. 1982 3M	12 S Wyoming‖	6	TFR
Yellowstone Co., MT 4 Sept. 1982	30 Aug. 1982 3N	9 N Wyoming‖	5	TFR
Jerome Co., ID 27 Aug. 1982	20 Aug. 1982 3O	12 W Utah 30 NW Arizona	7	TFR
Otoe Co., NE 25 May 1983	12 May 1983	30 S Texas‖	13	TFR

*AZ, Arizona; CO, Colorado; ID, Idaho; MT, Montana; NE, Nebraska; NM, New Mexico; UT, Utah; WY, Wyoming.

†Source 18 h previously.

‡T, temperature ⩾ 10 °C; F, front; R, rain.

§See B etc. in Fig. 3.

‖Trajectory at 850 mb; in the other instances 850, 900 and 1000 mb trajectories were the same.

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trajectories are given for periods up to 30 h. Thirty-five hours is the calculated maximum flight endurance of *Simulium* species [20]; of the insects possibly involved in transmission of VS virus *Simulium* have the longest flight endurance [21]. Representative trajectories are shown in Fig. 2(*a–d*).

A possible route for the spread of VS from 23 May to 4 September 1982 can be derived from the backward trajectories (Fig. 3). Spread would have been from northern Sonora, Mexico, to Arizona (18 May) and north-eastward to Albuquerque, New Mexico (14 June) and from western Sonora eastward over the Continental Divide to Chihuahua (30 May). From Albuquerque VS would have spread northwards to northern New Mexico and into southern Colorado (July and August). West of the Continental Divide spread from Arizona would have taken place north-eastwards to La Plata and Mesa Counties, Colorado and to Grand County, Utah (16–17 July) and to Uinta and Duchesne Counties, Utah (23 July). Spread would then have been to Sweetwater County, Wyoming, and eastwards across the Continental Divide to Jefferson and Adams Counties, Colorado, and to Converse County, Wyoming (2–3 August). In Wyoming the disease would have spread north to Bighorn County, Wyoming (11 August) and into Yellowstone County, Montana (30 August). The outbreaks in Idaho could have come from western Utah (20 August). The arrival of the 14 trajectories was accompanied by a front and rain in 12 instances and by a front alone in two.

VS in 1982–3

Subsequent spread of VS in 1982–3 apart from states already mentioned (Arizona, Colorado, Idaho, Montana, New Mexico, Utah and Wyoming) was found to be through movement of animals and VS was reported in Missouri, Washington, California, Kansas and Nebraska [1]. The last outbreak in this series was in Otoe County, Nebraska on 25 May 1983. Examination of backward trajectories for this location showed that at 850 mb the origin could have been west of San Antonio, Texas, 30 h before the arrival on 12 May (Table 1). However no VS had been reported from that area of Texas and this outbreak could have resulted from introduction of infection from previous outbreaks in Nebraska earlier in the year [1].

VS in 1983–4

There were three outbreaks of VS, New Jersey, in Colorado and Wyoming in August and September 1983 [1]. In early 1984 VS was found in Texas in cattle, which had been purchased from Durango, Mexico, where VS was present [1]. At the end of 1984 VS was diagnosed at Marfa, Texas [2]

VS in 1985

In 1985 outbreaks in southern New Mexico in May and in Arizona and central New Mexico in June were followed by outbreaks in Pueblo, Colorado and in north-western New Mexico and south-western Colorado in July ([2]; L. C. Vanderwagen, personal communications, 1986–7; M. A. Mixson, personal communications, 1987–8). An outbreak of VS was also reported in Sonora, Mexico in May (J. Mason, personal communication, 1989). Locations are shown in Fig. 1.

The outbreaks for which backward trajectories were determined and the

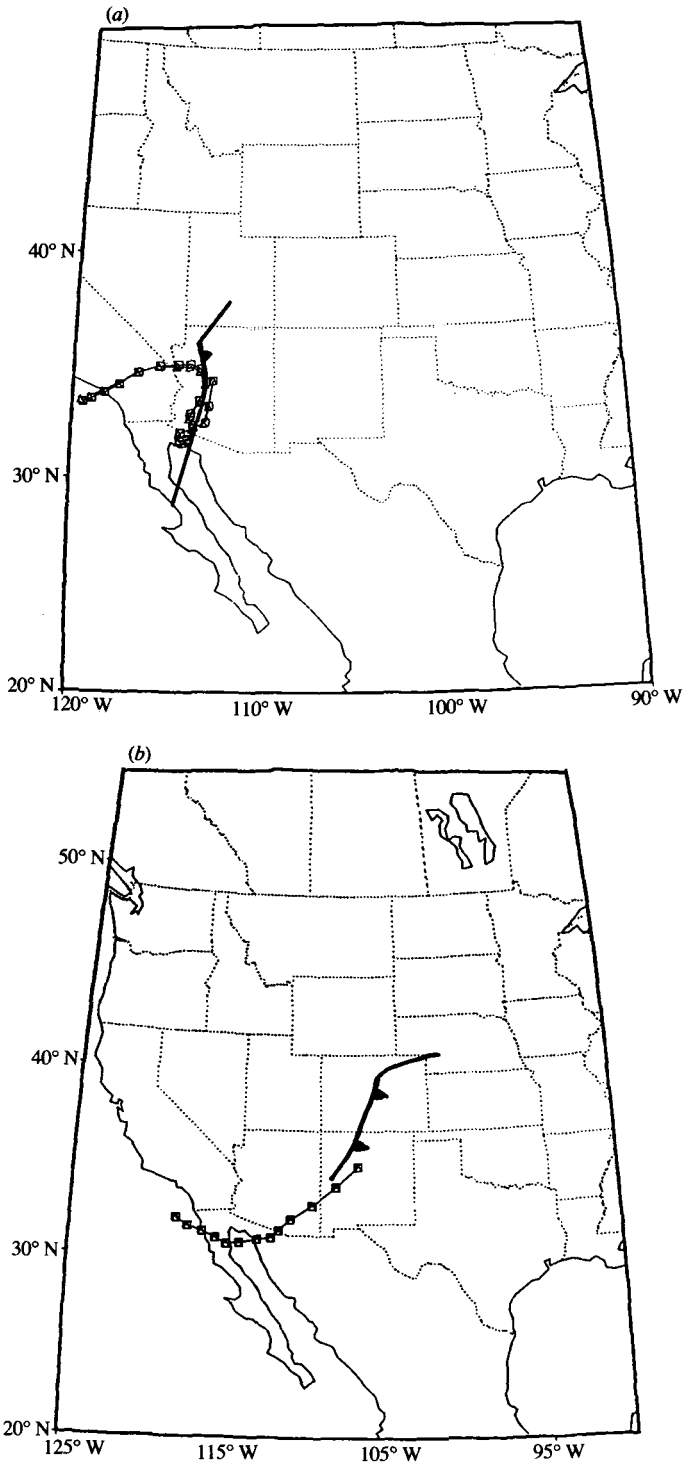

Fig. 2. Fronts and 6-hourly segments of backward trajectories at three levels (△, 850 mb; ○, 900 mb; □, 1000 mb) terminating at (*a*) Camp Verde, Arizona on 19 May 1982 at 00.00 GMT (17.00 MST 18 May 1982). (*b*) Albuquerque, New Mexico on 14 June 1982 at 18.00 GMT (11.00 MST). (*c*) Mesa Co., Colorado on 16 July 1982 at 18.00 GMT (11.00 MST). (*d*) Denver, Colorado on 3 August 1982 at 18.00 GMT (11.00 MST).

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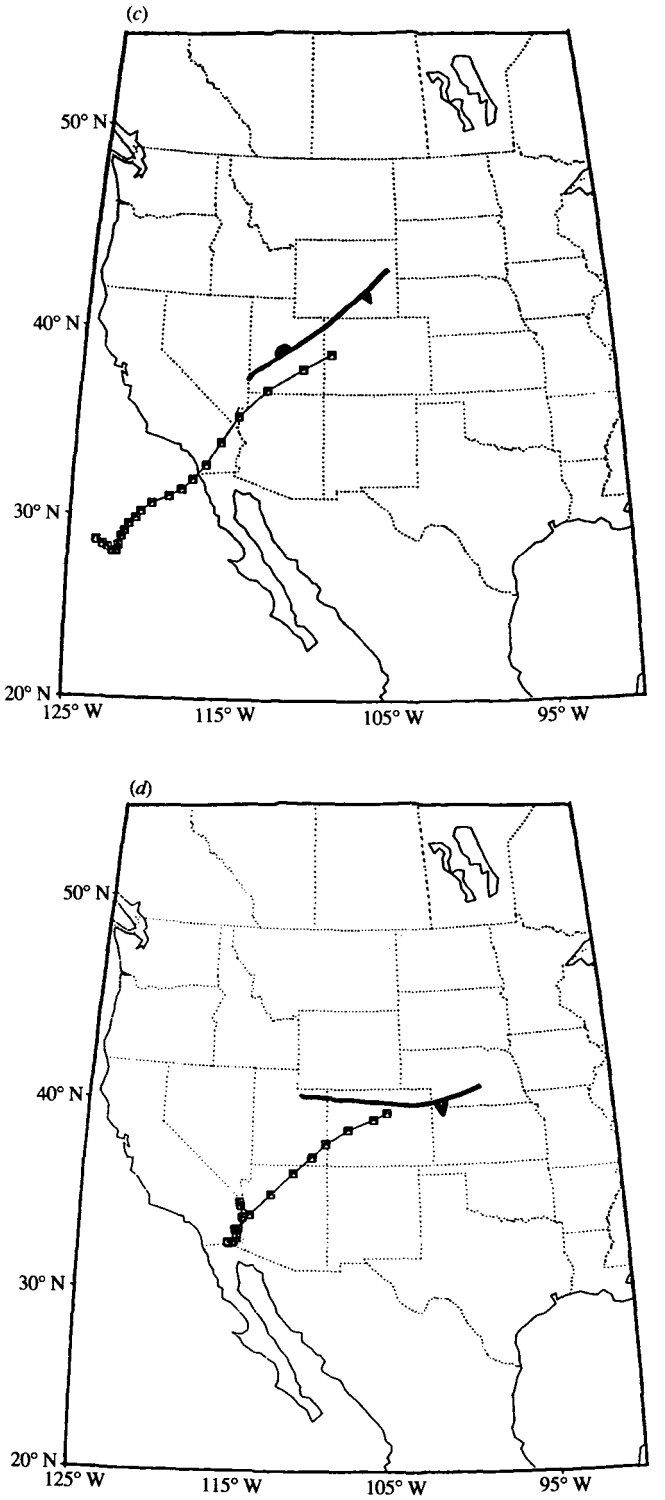

Fig. 2 (*c*, *d*). For legend see opposite.

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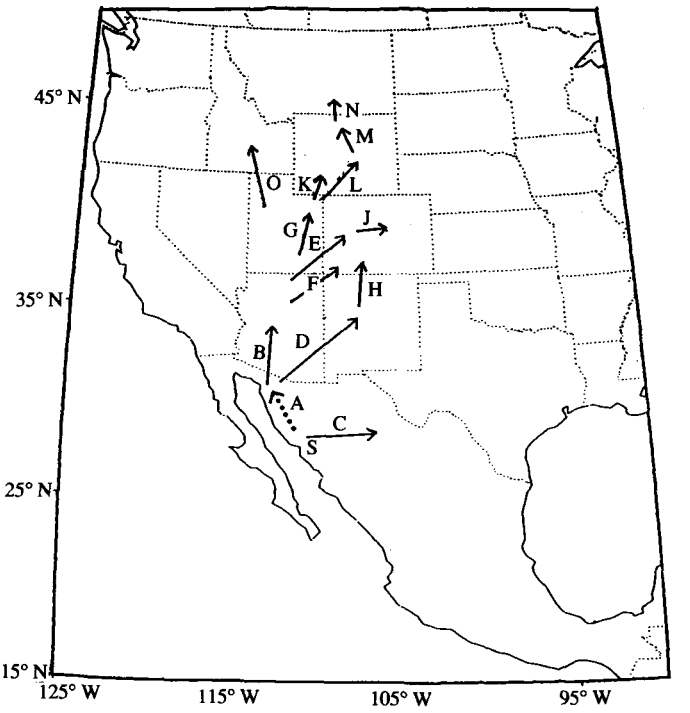

Fig. 3. Suggested spread of VS May to September 1982 based on analysis of backward trajectories. ⟶, spread based on trajectories; ⋯⟶, possible spread. A, Sonora, Mexico. B, Sonora to Camp Verde, AZ (18 May). C, Sonora to Chihuahua, Mexico (30 May). D, Sonora to Albuquerque, NM (14 June). E, Arizona to Mesa County, CO (16 July). F, Arizona to Durango, CO (16 July). G, Arizona to Uinta County, UT (23 July). H, New Mexico to S. Colorado (27 July). J, W. Colorado to C. Colorado (2–3 Aug.). K, NE Utah to Sweetwater County, WY (2–3 Aug.). L, NE Utah to Converse County, WY (2–3 Aug.). M, S Wyoming to Bighorn County, WY (11 Aug.) N, N Wyoming to Yellowstone County, MT (30 Aug.). O, Utah to Jerome County, ID (20 Aug.). S, Site of outbreak in Sonora (April)

possible sources are shown in Table 2. Two of the backward trajectories are illustrated in Fig. 4. The outbreaks in southern New Mexico in May and in Arizona in early June could have had their origins in northern Sonora, Mexico. The outbreak in Aztec could have come from Arizona at the end of June. Backward trajectories indicate that the outbreak in Pueblo could have come either from Arizona at the end of June or from northern New Mexico at the beginning of July; it could also have started as a result of introduction of animals. A possible route for the spread of VS in 1985 is shown in Fig. 5. Arrivals of trajectories were associated with passage of a front and rain, passage of a front or rain alone.

Fig. 4. Fronts and 6-hourly segments of backward trajectories at three levels (△, 850 mb; ○, 900 mb; □, 1000 mb) terminating at: (*a*) Los Cruces, New Mexico on 10 May 1985 at 18.00 GMT (11.00 MST). (*b*) Los Lunas, New Mexico on 27 May 1985 at 18.00 GMT (11.00 MST).

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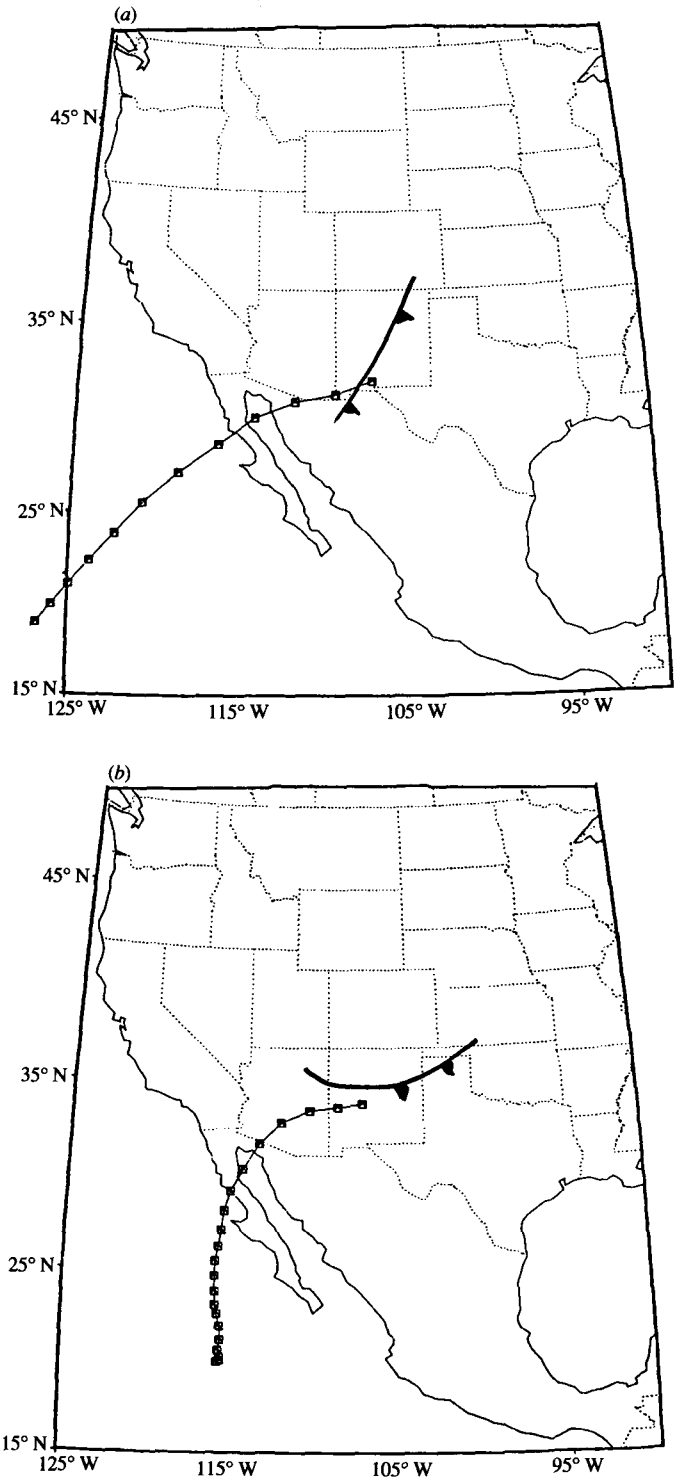

Fig. 4 (*a*, *b*). For legend see opposite.

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Table 2. *Source of backward trajectories at 850, 900 and 1000 mb up to 30 h previously together with weather conditions on arrival for outbreaks of vesicular stomatitis in western USA and in northern Mexico in 1985*

Location and date of disease	Date of arrival	Source at 850, 900 and 1000 mb		Interval (days)	Conditions
Los Cruces, NM* 15 May 1985 Fig. 4*a*	10 May 1985 5B§	12†	NW Sonora	5	TFR‡
Truth and Consequences, NM 25 May 1985	17 May 1985 5C	9	Los Cruces, NM	8	TR
Los Lunas, NM 1 June 1985 Fig. 4*b*	27 May 1985 5D	27	N Sonora, Mexico	5	TF
Fort Huachuca AZ 5 June 1985	31 May 1985	18	N Sonora, Mexico‖	6	TF
Pinetop, AZ 27 June 1985	21 June 1985 5E	12	SW Arizona	6	TF
Pueblo, CO 11 July 1985	25 June 1985 5F	8	Rio Arriba Co., NM	16	TF
	30 June 1985 5G	30	NE Arizona	11	TR
	7 July 1985 5J	12	Taos Co., NM	4	TF
Aztec, NM 15 July 1985	30 June 1985 5H	6	NE Arizona	15	TR

* AZ, Arizona; CO, Colorado; NM, New Mexico.
† Source 12 h previously
‡ T, temperature $\geqslant$ 10 °C; F, front; R, rain.
§ B, etc. in Fig. 5.
‖ Trajectory at 850 and 900 mb; in the other instances 850, 900 and 1000 mb trajectories were the same.

DISCUSSION

Trajectories and fingerprinting

The 1982 outbreaks and the 1985 outbreaks (apart from the outbreak in Pueblo), which were examined by backward trajectory analysis of winds, were outbreaks for which no evidence of introduction of virus by movement of animals was found ([1, 14]; G. A. Erickson, personal communication, 1988; M. A. Mixson, personal communication, 1988). The trajectories of the 1982 outbreaks can be considered in three groups: (i) the trajectories after the beginning of August from western Colorado and Utah into eastern Colorado, Wyoming and Idaho and from Wyoming into Montana, (ii) the trajectories from Arizona and New Mexico into Colorado and Utah and (iii) the trajectories from Sonora to Chihuahua, Arizona and New Mexico.

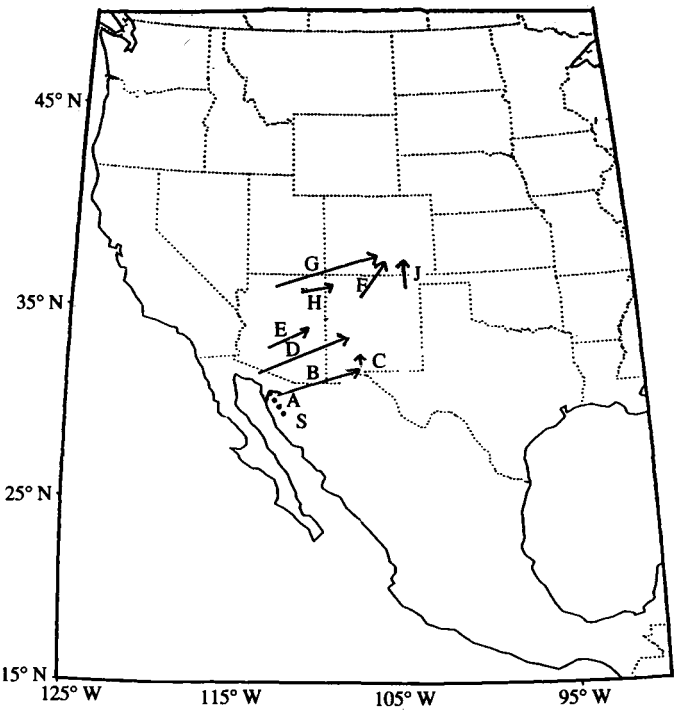

Fig. 5. Suggested spread of VS May to July 1985 based on analysis of backward trajectories. ⟶, Spread based on trajectories; ⋯⇢, possible spread. A, Sonora, Mexico. B, Sonora to Los Cruces, NM (10 May). C, Los Cruces to Truth and Consequences, NM (17 May). D, Sonora to Los Lunas, NM (27 May). E, Arizona to Pinetop, AZ (21 June). F, New Mexico to Pueblo, CO (25 June). G, NE Arizona to Pueblo, CO (30 June). H, NE Arizona to Aztec, NM (30 June). J, New Mexico to Pueblo, CO (7 July). S, Site of outbreak in Sonora (May).

Trajectories of the first group could be tracked back to areas where disease was occurring at the time of the trajectory. The T1 fingerprints of isolates associated with this group are similar or with 2 or 3 spots difference [10]. The last outbreak of VS in Wyoming and Montana before 1982 due to the New Jersey serotype was in 1949 [22]. Investigations after the 1949 outbreak in Wisconsin showed that, although neutralizing antibody persisted for 7 years, attempts to isolate virus were unsuccessful [6]. Persistence in Wyoming and Montana from 1949–82 in animals or on pasture would therefore seem unlikely. Persistent infection would lead to random evolution of virus, whereas the fingerprints of the isolates tested were genetically homogeneous [10, 12]. As there was no evidence of introduction by animal movement and in view of the correlation between fingerprinting and trajectory analysis, spread by carriage on the wind can be considered feasible.

No fingerprints were done on isolates of the second group. The trajectory of the outbreaks in Archuleta and Alamosa Counties gave a source in New Mexico, where disease was present. The trajectories of the other outbreaks gave sources in Arizona, where disease had been reported in May and July. Fingerprinting was also not done on isolates of the third group. The trajectories for the outbreaks in Arizona and New Mexico could be tracked back to the northern part of Sonora,

whereas the outbreak in that state in April was in the southern part of the state (J. Mason, personal communication, 1989). Examination of weather maps shows that infection could have been taken on the wind from the south to the north of Sonora and movement of infected animals is also a possibility.

In 1985 the trajectories of early outbreaks in New Mexico and Arizona could be tracked back to northern Sonora. VS had been reported in early May in central Sonora (J. Mason, personal communication, 1989). The later outbreaks in Arizona, New Mexico and Colorado could be traced to areas, where disease had been observed. The fingerprints of the isolates from Sonora and Chihuahua in Mexico were similar to or differed by two spots from fingerprints from isolates in New Mexico, Arizona or Colorado [10]. The findings from the trajectory analysis would appear consistent with those from T1 fingerprint analysis.

The fingerprint of the isolate from Nebraska in May 1983 was identical or similar to the fingerprints of the 1982 isolates [10]. The backward trajectory analysis showed a possible source near San Antonio, Texas; however no disease was reported from that area [1]. Fingerprints of isolates from cattle imported into Texas in early 1984 were similar to fingerprints of isolates collected in an outbreak in Mexico the previous month [10].

The 1982 US isolates differed by 20 spots from the 1984 Texas isolate and by 22 spots from the 1985 US and Mexico isolates [10]. The Texas isolate differed by 8 spots from the 1985 isolates. All isolates from these years were considered to constitute a single T1 fingerprint group, which extended as far south in Mexico as Vera Cruz [10–12]. An isolate from Vera Cruz in 1982 differed by 12 spots from the 1982 US isolates [10]. It was strongly suggested on the basis of the fingerprint findings and on epidemiological observations that Mexico might be the source of the VS virus isolated in the USA [10, 12]. The findings of the trajectory analyses would support this suggestion in respect of the northward spread, where animal movement was not involved but carriage of infection on the wind was a possibility.

Carriage on the wind of particles and insects

Analysis through backward trajectories of winds is applicable to outbreaks where virus is carried as airborne particles or where infection is carried by infected insects on the wind.

Horses and cattle may be infected with VS by virus aerosols and infected animals emit aerosols in amounts which can infect man or animals in close contact [5, 6]. The amount of virus given out as an aerosol and the amount of virus required to infect susceptible animals are not known. However an estimate can be made of the amount of virus given out per minute from a modification of Pasquill's formula [23] and by assuming that a susceptible animal downwind will inhale one infectious particle in a day:

$$V = \frac{W \times D \times H \times A \times 10^3}{2{\cdot}8 \times I},$$

(V, amount of virus given out per minute; W, windspeed as m s^{-1}; D, distance in km; H, height of plume in m; A, angle of plume in degrees; I, volume of air inhaled in a day by one cattle beast in m^3). With $W = 10$ m s^{-1}, $D = 200$ km, $H = 300$ m, $A = 10^0$ and $I = 150$ m^3, a value of $1{\cdot}4 \times 10^7$ is obtained. This would give

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$1{\cdot}4 \times 10^5$ per min per cattle beast if a herd of 100 cattle were the source. This amount is of the same order as that emitted by a pig infected with foot-and-mouth-disease virus and a thousandfold greater than that emitted by cattle and sheep [24]. With amounts more than 10^8 per animal per day one would expect far more infection in the field under close contact than was found in 1982 [16, 18, 25] and so this method of airborne spread can be considered unlikely.

The New Jersey serotype of VS virus has been isolated from non-biting flies, Anthomyidae (Colorado), *Hippelates* (Colorado), *Musca autumnalis* and *M. domestica* (Colorado), and from biting flies, *Simulium* (Colombia and Colorado), *Culicoides* (Colorado), *Culex nigripalpus* (Guatemala) and *Mansonia indubitans* (Ecuador) [8]. The non-biting flies feed on the oral, nasal and lachrymal secretions or on lesions on the feet and teats and transmit the virus mechanically from animal to animal or through contaminated foodstuffs [8]. *M. domestica* has been observed to fly for distances of 32 km and Muscidae have been caught at a height of 660 m [21]. These insects could be responsible for spread within herds but are unlikely to be responsible for spread over long distances.

During the 1982 outbreak VS virus was isolated from *Simulium* and *Culicoides* [15, 16]. Virus of low titre was usually isolated, so transmission would be mechanical. Virus of higher titre was isolated from a pool of *S. vittatum* and *S. bivittatum* indicating possible biological transmission [15]. *Simulium* and *Culicoides* may be carried on the wind for distances of up to 600–700 km at heights up to 1·5 km and for a duration of 24–35 h [26, 27]. These insects have been implicated in the transmission on the wind of onchocerciasis (*Simulium*, [28]) and bluetongue, African horse sickness and Akabane Disease (*Culicoides*, [26, 29]). The minimum temperature for flight for *C. variipennis* is 10 °C [30] and temperatures of 10 °C and higher were found at heights of up to 2700–3500 m at the times of suggested trajectories in 1982 and 1985. *Simulium* and *Culicoides* were caught at heights up to 2200 m in 1982 [16].

The reports of disease along valleys, wet wooded areas and in pastures are consistent with the behaviour of *Simulium* [15]. The distribution of *S. vittatum* from Mexico to the Canadian border makes it together with *S. bivittatum* the most likely species for virus transmission ([15, 31]; M. M. Galloway, personal communication, 1987).

However in infected ruminants there may be no viraemia or only a viraemia of short duration and low titre [6–8, 32] and the source of virus at the time of the insect bite has been thought to be elsewhere [6, 8]. Monath and colleagues [8] attributed the source of virus to lesions in and around the mouth, on the teats and on the feet. Other sites could be the skin, especially the predilection sites for *Simulium* and *Culicoides* along the belly and the back and on the ears [33, 34]. Heiny [35] reported lesions on the legs as far up as the hocks, the belly, prepuce and ears. Foot-and-mouth-disease virus (titres $> 10^4$ ID_{50} per g) has been isolated from the skin even when no lesions are visible [36]. Pathogenesis studies on vesicular diseases have shown that lesions develop as a result of trauma [37–39] and insect bites on the skin could lead to microscopic lesions. Another possible source of virus could be monocytes, which have been found at the site of lesions and have been shown to support the multiplication of VS virus and be responsible for the clearance of virus from the circulation [40–42]. Of the insects, from which

VS virus has been isolated, *Simulium* would appear to be the most likely to transmit the virus over long distances; however further investigations on biological and mechanical transmission would be required [15].

Epidemic spread and conclusions

Spread by movement of cattle and horses occurred during the 1982–6 epidemic [1]. Evidence was found of continuing infection in dairy herds, feedlots and on horse ranches for 1–2 months [1, 13, 18]. There was inapparent infection, shown by the development of antibodies, in domestic and wild ruminants [16, 43]. These findings would explain some of the spread during the epidemic and the persistence of virus during the winter months of 1982–3. The findings from the trajectory analyses of outbreaks in Colorado, Wyoming and Montana in 1982 together with the results of fingerprinting indicate that spread by carriage on the wind was feasible. Infected insects carried on warm winds would remain aloft until the warm air met a cold front, where convergence would lead to landing of insects and their concentration [17]. Such a method of spread could explain the movement of disease during the epidemic, where evidence of introduction of infected animals was absent and where there were outbreaks at long distances without intermediate outbreaks as well as the occurrence of many outbreaks at the same time.

If spread is to occur through carriage on the wind of infected insects such as *Simulium*, a number of factors must coincide: (i) the presence of virus in animals at source, (ii) insects in sufficient numbers at source, (iii) temperature $\geqslant 10$ °C at the beginning and during flight to the height at which the insects are flying and suitable winds meeting cold fronts where the insects land, (iv) susceptible animal population at the point of arrival.

In 1982 and 1985 VS, New Jersey serotype, was reported in Sonora in April and May respectively. From 1975–81 and in 1983, 1986 and 1987 no disease was reported in Sonora; in 1984 disease was reported in pigs in January in the south of the state ([5]; J. Mason, personal communication, 1989). Thus for the years when disease was reported in Arizona and New Mexico, disease was present in Sonora at a time when insects might be active. The insect population at that time in Sonora is not known; minimum temperatures during April and May were 14 °C and there were winds which met cold fronts as they moved northwards into Arizona and New Mexico. The cattle and horse population in Arizona and New Mexico was susceptible because no outbreaks of VS had been reported since 1972. Once VS had entered western USA, there were sources of disease and reports of large swarms of *Simulium* as well as catches of *Simulium* and *Culicoides*, from which virus was isolated [1, 14–16]. Temperatures were greater than 10 °C and there were winds meeting cold fronts. The failure of the 1985 epidemic to move further north in July 1985 could have been due to the presence of cold fronts during the first half of the month.

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REFERENCES

1. Jenney EW, Erickson GA, Snyder ML. Vesicular stomatitis outbreaks and surveillance in the United States January 1980 through May 1984. Proc US Anim Hlth Assoc 1984; **88**: 337–49.
2. Hall AE. Emergency programs progress report. Proc US Anim Hlth Assoc 1985; **89**: 229–32.
3. Jenney EW. Vesicular stomatitis in the United States during the last five years (1963–1967). Proc US Livestock Sanit Assoc 1967; **71**: 371–85.
4. Jenney EW, Erickson GA, Buisch WW, Stewart WC, Mixson MA. Surveillance for vesicular stomatitis in the United States 1972 through 1979. Proc Am Assn Vet Lab Diagn 1980; **23**: 83–9.
5. Mason J. Vesicular stomatitis in Mexico. Vesicular stomatitis field studies in Mexico. In: Proceedings of an International Conference on Vesicular Stomatitis. Mexico City: Mexico–United States Commission for the Prevention of Foot-and-Mouth Disease, 1984: 118–47, 276–297.
6. Hanson RP. Vesicular stomatitis. In: Gibbs EPJ ed. Virus diseases of food animals. London: Academic Press, 1981: 517–39.
7. Mason J. The epidemiology of vesicular stomatitis. Bltn Centro Panamericano Fiebre Aftosa 1978; **29–30**: 35–53.
8. Monath TP, Webb PA, Francy DB, Walton TE. The epidemiology of vesicular stomatitis–new data, old puzzles. In: St George TD, Kay BH, Blok J, eds. Proceedings Fourth Symposium Arbovirus Research in Australia. Brisbane: CSIRO, 1986: 193–9.
9. Jonkers AH. The epizootiology of the vesicular stomatitis virus: a reappraisal. Am J Epidemiol 1967; **86**: 286–91.
10. Nichol ST. Molecular epizootiology and evolution of vesicular stomatitis virus New Jersey. J Virol 1987; **61**: 1029–36.
11. Nichol ST. Genetic diversity of enzootic isolates of vesicular stomatitis virus New Jersey. J Virol 1988; **62**: 572–9.
12. United States Department of Agriculture. Vesicular stomatitis virus fingerprinting. For Anim Dis Rep 1988; **16–1**: 8–11.
13. Thurmond MC, Ardans AA, Picanso JP, McDowell T, Reynolds B, Saito J. Vesicular stomatitis virus (New Jersey strain) infection in two California dairy herds: an epidemiologic study. J Am Vet Med Assoc 1987; **191**: 965–70.
14. McDaniel HA. History of vesicular stomatitis in the United States. In: Proceedings of an International Conference on Vesicular Stomatitis. Mexico City: Mexico–United States Commission for the Prevention of Foot-and-Mouth Disease, 1984: 159–67.
15. Francy DB, Moore CG, Smith GC, Jakob WL. Taylor SA, Calisher CH. Epizootic vesicular stomatitis in Colorado, 1982: isolation of virus from insects collected along the northern Colorado Rocky Mountain front range. J Med Entomol 1988; **25**: 343–7.
16. Walton TE, Webb PA, Kramer WL et al. Epizootic vesicular stomatitis in Colorado, 1982: epidemiologic and entomologic studies. Am J Trop Med Hyg 1987; **36**: 166–76.
17. Sellers RF, Maarouf AR. Impact of climate on western equine encephalitis in Manitoba, Minnesota and North Dakota, 1980–1983. Epidemiol Infect 1988; **101**: 511–35.
18. Webb PA, Monath TP, Reif JS, et al. Epizootic vesicular stomatitis in Colorado, 1982: epidemiologic studies along the northern Colorado front range. Am J Trop Med Hyg 1987; **36**: 183–8.
19. Olson MP, Oikawa KK, Macafee AW. A trajectory model applied to the long-range transport of air pollutants. Atmospheric Environment Service, Long Range Transport of Air Pollutants 78–4, 1978: 24.

20. Hocking B. The intrinsic range and speed of flight of insects. Trans R Entomol Soc Lond 1953; **104**: 223–345.
21. Johnson CG. Migration and dispersal of insects by flight. London: Methuen, 1969: 763.
22. Hanson RP. The natural history of vesicular stomatitis. Bacteriol Rev 1952; **16**: 179–204.
23. Pasquill F. The estimation of the dispersion of windborne material. Met Mag 1969; **90**: 33–49.
24. Donaldson AI, Lee M, Gibson CF. Improvement of mathematical models for predicting the airborne spread of foot-and-mouth disease. In: Boehm G, Leuschner RM, eds. Advances in aerobiology. Basel: Birkhauser Verlag 1987: 351–5.
25. Reif JS, Webb PA, Monath TP, et al. Epizootic vesicular stomatitis in Colorado, 1982; infection in occupational risk groups. Am J Trop Med Hyg 1987; **36**: 177–82.
26. Sellers RF. Seasonal variations in the spread of arthropod-borne disease agents of man and animals: implications for control. Phil Trans R Soc Lond B 1983; **302**: 485–95.
27. Rempel JG, Arnason AP. An account of three successive outbreaks of the black fly, *Simulium arcticum*, a serious livestock pest in Saskatchewan. Sci Agric 1947; **27**: 428–45.
28. Cheke RA, Garms R. Reinfestations of the southeastern flank of the Onchocerciasis Control Programme area by windborne vectors. Phil Trans R Soc Lond B 1983; **302**: 471–84.
29. Murray MD, Akabane epizootics in New South Wales: evidence for long-distance dispersal of the biting midge *Culicoides brevitarsis*. Aust Vet J 1987; **64**: 305–8.
30. Nelson RL, Bellamy RE. Patterns of flight activity of *Culicoides variipennis* (Coquillett) (Diptera: Ceratopogonidae). J Med Entomol 1971; **8**: 283–91.
31. Crosskey RW. Geographical distribution of Simuliidae. In: Laird M, ed. Blackflies. London: Academic Press, 1981: 57–68.
32. Gay J. Vesicular stomatitis – pathogenesis studies. In: Proceedings of an International Conference on Vesicular Stomatitis. Mexico City: Mexico–United States Commission for the Prevention of Foot-and-Mouth Disease, 1984; 312–33.
33. Eichler DA. Studies on *Onchocerca gutterosa* (Neumann 1910) and its development in *Simulium ornatum* (Meigen 1818). II. Behaviour of *S. ornatum* in relation to the transmission of *O. gutterosa*. J Helminthol 1971; **45**: 259–70.
34. Mellor PS. Studies on *Onchocerca cervicalis* Raillet and Henry 1910: IV. Behaviour of the vector *Culicoides nubeculosus* in relation to the transmission of *Onchocerca cervicalis*. J Helminthol 1974; **48**: 283–8.
35. Heiny E. Vesicular stomatitis in cattle and horses in Colorado. North Amer Vet 1945; **26**: 726–30.
36. Gailiunas P, Cottral GE. Presence and persistence of foot-and-mouth disease virus in bovine skin. J Bacteriol 1966; **91**: 2333–7.
37. Shahan MS, Frank AH, Mott LO. Studies of vesicular stomatitis with special reference to a virus of swine origin. J Am Vet Med Assoc 1946; **108**: 5–19.
38. Mann JA, Burrows R, Goodridge D. Mild and sub-clinical infections with swine vesicular disease virus. Bull Off Int Epizoot 1975; **83**: 117–22.
39. Skinner HH, Knight EH. Environmental factors influencing the response of guinea-pigs to modified strains of foot-and-mouth disease virus. Bull Off Int Epizoot 1964; **61**: 1523–43.
40. Seibold HR, Sharp JB. A revised concept of the pathologic changes of the tongue in cattle with vesicular stomatitis. Am J Vet Res 1960; **21**: 35–51.
41. Edelman R, Wheelock EF. Specific role of each human leukocyte type in viral infections. I. Monocyte as host cell for vesicular stomatitis virus replication in vitro. J Virol 1967; **1**: 1139–49.
42. Tesh RB, Peralta PH, Johnson KM. Ecologic studies of vesicular stomatitis virus. II. Results of experimental infection in Panamanian wild animals. Am J Epidemiol 1970; **91**: 216–24.
43. Webb PA, McLean RG, Smith GC *et al.* Epizootic vesicular stomatitis in Colorado, 1982: some observations on the possible role of wildlife populations in an enzootic maintenance cycle. J Wildl Dis 1987; **23**: 192–8.