Overview

Following the dramatic spread of myxomatosis in Australia early in 1951, an illegal introduction for rabbit control was made on an estate near Paris in June 1952. The strain of virus used (‘Lausanne’) was recently derived from its *Sylvilagus* host in Brazil. It led to the establishment of the disease in France. Over the next decade myxomatosis spread to most of the countries of Europe in which rabbits are found, producing very high mortalities.

The public reaction to myxomatosis in France was dominated by deep concern by rabbit breeders for the safety of their domestic rabbits and by chasseurs for the destruction of an important hunting animal, the wild rabbit. On the other hand, foresters and most farmers welcomed the destruction of a major pest. Vaccination of domestic and wild rabbits was practised on a large scale.

The Lausanne strain was highly lethal and the skin lesions were much more protuberant than those produced by the virus used for the Australian introduction. Less virulent strains were recognized two years after its introduction, and tests nine and fifteen years later showed that a variety of strains of differing virulence were present, the percentage of highly virulent strains decreasing progressively to 2% by 1962. In 1980 a ‘non-myxomatous’ or ‘respiratory’ form of myxomatosis was observed in commercial rabbitries; it soon became the commonest form and was thought to be transmitted by close contact.

Initially the populations of wild rabbits were decimated, and even with the reduction in virulence of the virus and the increased resistance of the rabbits, by the 1990s myxomatosis appeared to have produced reductions in the wild rabbit populations to somewhat less than half those found before 1953.

Introduction into France

The dramatic outbreak of myxomatosis among rabbits in Australia in early 1951 received extensive media coverage in Europe. The first international responses to this demonstration of its effectiveness for rabbit control occurred in France.

*Enquiries from the Institut Pasteur, Paris, January 1952*

On 22 January 1952 Dr G. Remaudière, of the Service de Parasitologie vegetale of the Institut Pasteur, wrote to Francis Ratcliffe, the head of the Wildlife Survey Section of CSIRO, requesting reprints on myxomatosis, a specimen of the virus and details on its cultivation, and saying, amongst other things:

Les lapins constituant dans certaines parties de la France, un véritable fléau, l’Institut Pasteur désirerait entreprendre la lutte biologique contre ce Rongeur et essayer de développer une épidémie de myxomatose en
Sologne. Cette région infestée de lapins semble, à priori, favorable à l’expansion de la maladie, car les Culicidés et Simulidés sont abondants.

Ratliffe passed the letter to his colleague Fenner, who on 15 February 1952 sent Remaudière two vials of freeze-dried myxoma virus and full particulars of the method of preparation of large quantities for inoculation campaigns. Receipt of the letter and virus was acknowledged on 29 February, but no further information about the use of the virus was received in Australia or could be found in a recent search of archival sources in France.

Release of myxoma virus on Maillebois Estate, June 1952

Dr P.F. Armand Delille (Fig. 9.1) was a distinguished physician and bacteriologist who was 77 years old when he read in the Paris papers of the great epizootics of myxomatosis in Australia in 1950–51, and conceived the idea of eliminating wild rabbits from his estate at Chateau Maillebois (Eure-et-Loire) by the use of this virus. Lockley describes his visit to Maillebois in December 1953 in the following terms (Lockley, 1964):

At that moment Dr Delille was a very worried man; proceedings against him had been begun by hunting and sporting interests in France, and he was engaged with his legal advisers in Paris preparing to defend his actions in the law courts. A test case was being brought against him, with the financial backing of the hunting clubs of France, by the local owner of a domestic rabbitry in which the rabbits had all died of myxomatosis ...

The Chateau Maillebois is a striking turreted medieval house lying in a beautiful wooded estate of 600 acres with a farm and small river, the whole enclosed with a high stone wall. This wall is broken only by certain entrance gates which had been rendered rabbit-proof before the introduction of the virus. Thousands of wild rabbits were devouring the farm crops and killing the tender forest trees, Dr Delille’s son told us, when in June, 1952, two wild-caught rabbits were inoculated with myxoma virus. In six weeks about 98% of the wild rabbits were dead, but none of the domestic rabbits in the hutches on the estate was affected.

In October 1952, the disease was identified from a corpse picked up near Rambuoillet, the residence of the President of France, fifty kilometres from Maillebois.

The disease at Rambouillet was identified as myxomatosis by Jacotot and Vallée (1953), but at the time its source was unknown to the authorities or the public. Concerning the escape from Maillebois, Delille did not
believe that the virus had been carried by mosquitoes, because his own domestic rabbits had not been affected. He thought that perhaps other landowners had broken in and captured diseased rabbits for distribution on their properties. About a year later, by which time some 35% of domestic rabbits and an estimated 45% of wild rabbits in France had died of myxomatosis, Delille made a public statement. On 14 June 1953 and again on 14 October 1953, he read papers to the Académie d’Agriculture, claiming that the method should be used systematically for rabbit control (Delille, 1953). At this time, one of us (F.F.) was in Paris, and could not but be amused by the controversy then raging in Le Temps about the height that a rabbit could jump, because this had been proposed as the way by which an infected rabbit had escaped from Maillebois.

While agriculturalists and especially foresters supported Delille, and were ultimately to award him a gold medal (Fig. 9.2), he was vigorously denounced by the powerful hunting organizations. The Ministry of Agriculture was in a difficult position, because in addition to its duties in relation to agriculture and forestry, it was responsible for the Conseil Supérieure de La Chasse, which derived its income from gun licence revenue paid by sportsmen who were primarily interested in hunting rabbits. Bills were introduced into the French Assembly to make the introduction and use of myxomatosis illegal, but it was too late – the disease was established and spreading. With the financial support of the hunting clubs of France, a test case was brought against Delille by the owner of a local domestic rabbitry, all of whose rabbits had died of myxomatosis. The case failed on a technicality – it could not be proved that the virus had been introduced directly into the affected rabbitry by Delille (as indeed it had not).

The virus that Delille had used differed from those used previously for introductions into European rabbits, which were derived from the Moses strain and had been extensively passaged in laboratory rabbits. It was sent to Delille by his friend Professor Hauduroy, Director of the Centre de Collection de Types microbien in Lausanne, Switzerland. Dr G. Bouvier, Director of Institut Galli-Valerio in Lausanne, has described the origin of this strain (Bouvier, 1954), which was derived from the Sylvilagus reservoir in Brazil with at most six passages in domestic rabbits before being sent to Delille. It has been called ‘Lausanne’ in scientific papers since a description of its properties by Fenner and Marshall (1957).

Attitude to Rabbits in France

Myxomatosis was a subject of great public interest in France, because there was a large commercial rabbit-breeding industry, many people had back-yard hutches (‘clapiers’) and there was a large and influential hunting fraternity for whom the wild rabbit was an important resource. On the other hand, foresters and farmers were very much aware of the importance of rabbits as an agricultural pest (Fig. 9.3). They shared with wild boars the distinction of being regarded as the major vertebrate pest, and in 1952, before the advent of myxomatosis, rabbit damage to agriculture and forestry had reached an estimated annual cost of 1000 million francs and 88 of the 90 départements in France had declared the rabbit a pest (Siriez, 1957).

The French Game Act of 1844, with later amendments, protects wild rabbits as game; predators which attack rabbits or
game birds, such as foxes, stoats and weasels, are regarded as vermin. There is an open season for hunting rabbits between early September and early January, with possible extension to 31 March. In 1952, the year before the introduction of myxomatosis, 1,850,000 shooting permits were issued, of which some 80% were held by persons who were primarily rabbit hunters. The importance of adequate numbers of wild rabbits to these enthusiastic chasseurs is obvious (see Table 9.1, below), as well as their significance for the supporting industries indicated in Fig. 9.3. The major hunting organizations, the Conseil Supérieur de la Chasse and the St Hubert Club de France, made prolonged and vigorous protests about the destruction of rabbits by myxomatosis, and supported such counter-measures as the vaccination of wild rabbits with fibroma virus, the introduction of cottontails from the United States, and the introduction of resistant rabbits from Australia, all to no avail.

Besides the importance of wild rabbits for hunters, there was a large domestic rabbit industry. In 1950 some 140 million domestic rabbits were produced and consumed annually in France, and many retired workers were partly dependent upon rabbit raising for their livelihood. Because mosquitoes were major vectors and myxomatosis was sweeping through the wild rabbit population, many outbreaks of the disease occurred among domestic rabbits in the early 1950s.

Short books on myxomatosis soon appeared (Radot and Lépine, 1953; Virat-Pilet, 1954) and a comprehensive two-volume monograph was published in 1972–73 (Joubert et al., 1972, 1973). The spread of myxomatosis in France prompted the organization of an International Symposium on Myxomatosis by l’Office Internationale des Epizooties in Paris in November 1953, and there were special sections on myxomatosis at the International Veterinary Congress in Stockholm in August 1953 and at the International Congress of Microbiology in Rome in September 1953.

**Official Action on Myxomatosis**

A Central Service for the Fight Against Myxomatosis was set up by the Conseil Supérieur de la Chasse on 16 July 1953, responsible to Dr F. Barthélémy, Engineer in Charge of Water and Forestry. It worked in close collaboration with the Veterinary Service, the Laboratory of Veterinary Research and the Institut Pasteur. Details of legislation providing for the control of myxomatosis are set out in the monograph.
The French Rural Code (RC) and Penal Code (PC) severely penalize deliberate importation and spread of infectious diseases of domestic and wild animals (PC articles 452, 454–1, 31 October 1955). Some articles of the Rural Code authorize the destruction of animal pests (RC articles 393–395) and specify the methods that can be used. Rabbits can be shot, trapped or poisoned. Sanitary regulations of rabbit farms are strictly policed, and farmers are required to maintain a register containing all information on diseases of their stock and vaccinations carried out.

No immediate official action was taken when myxomatosis was first recognized in October 1952. However, after the rapid spread of the disease in Spring 1953, decrees were published by the Ministry of Agriculture on 27 May 1953 and 27 June 1953. These made myxomatosis a notifiable disease and prohibited the movement of rabbits in an infected area. Appropriate measures were specified for the isolation and disinfection of domestic rabbitries, dead animals being incinerated and their remains buried in quicklime. It was further required that the sanitary status of rabbit farms had to be published fortnightly in the National Sanitary Bulletin (Ministerial decree of 9 July 1953). Persons guilty of the voluntary propagation of an epizootic were liable to imprisonment for a period of one to five years. In the case of wild rabbits, areas at risk had to be indicated by notices bearing the words ‘Myxomatose, maladie contagieuse du lapin’, and all rabbits within the area were supposed to be destroyed.

However, strict as these regulations seem to be, it was impossible to eliminate the disease, and periodically outbreaks continue to occur among wild rabbits, and sometimes spread from them to domestic rabbits which are not protected by vaccination or insect-proof screening.

**Clinical Features of Myxomatosis as Seen in France**

Myxomatosis in France was initiated by the inoculation of two wild rabbits with the Lausanne strain of myxoma virus; no further inoculations of virulent virus were ever made in the field. Laboratory studies in Australia showed that the Lausanne strain produced a disease (Fig. 9.4A) with more protuberant lesions than those caused by the Standard Laboratory Strain, initially used in Australia for rabbit control. In experiments conducted in Australia, challenge infections of rabbits with increased innate resistance showed that the Lausanne strain was also

![Fig. 9.4. Clinical signs of myxomatosis caused by strains of myxoma virus found in France during the early stages of the epizootic in France. (A) Lesions produced by the Lausanne strain. Rabbit photographed 10 days after inoculation; it died on the twelfth day. The primary lesion on the flank was very large, protuberant and deep purple in colour. The head was oedematous (‘leonine facies’) and the eyes completely closed with a profuse conjunctival discharge. The secondary lesions were also raised, purple in colour and not demarcated from the surrounding skin. (B) Lesions produced by the Loiret 55 strain. The photograph was taken on the 25th day and the rabbit died the next day. About one-third of rabbits inoculated with this strain recovered. The primary lesion was very large and exuded serum. There were numerous secondary lesions all over the body, the eyes were completely closed and the ears hung down because of numerous lesions on the pinnae. From Fenner and Marshall (1957), with permission.](image-url)
more virulent than the Standard Laboratory Strain, in that the case-fatality rate was about 99% when the Standard Laboratory Strain was associated with a case-fatality rate of 60–80%.

Attenuated strains of myxoma virus were first recognized in 1955, in the département of Loiret (Jacotot et al., 1955; Fig. 9.5). The lesions produced by this strain (‘Loiret 55’) retained the protuberance characteristic of the Lausanne strain lesions (Fig. 9.4B) but the disease evolved more slowly. In more extensive tests the case-fatality rate was 65% and the mean survival time 33.1 days, with a range of 19 days to recovery. It was later designated as the prototype European strain of Grade IV virulence.

Respiratory or non-myxomatous myxomatosis
In addition to the increase in the occurrence of attenuated strains characterized by the usual protuberant skin lesions but lower case-fatality rates (see below), in 1980 French scientists observed for the first time a ‘respiratory or non-myxomatous’ form of the disease (Brun et al., 1981b; Joubert et al., 1982). This syndrome was characterized by a longer incubation period (1–3 weeks), swelling of the eyelids accompanied by severe purulent conjunctivitis, genital lesions, and prominent nasal lesions accompanied by lacrimation and a mucopurulent nasal discharge (Fig. 9.6; see pp. 97 and 104). There were often pink or red spots on the ears, but no nodular skin lesions (Arthur and Louzis, 1988). Its epidemiology is described below (p. 220).

Although initially described in domestic rabbits raised by intensive husbandry and vaccinated with the SG33 attenuated myxoma virus vaccine (Chantal, 1981), this syndrome was also seen among rabbits kept under traditional husbandry and sometimes in wild rabbits. It was often accompanied by sterility and the abandonment of litters by farmed does.

The Spread of Myxomatosis in France
As commonly occurs when a dramatic ‘emerging’ disease breaks out, a number of fantastic stories about its mode of spread circulated during the early days of the French epizootic (Lockley, 1953). The consumption of grass that might have been contaminated by myxomatous wild rabbits
was thought to transfer myxomatosis to hutch rabbits, and motor cars which ran over infected rabbits were blamed for moving the disease to new districts. It was commonly reported that there was a black market for myxomatous rabbits, up to 5000 francs being paid by landholders anxious to get rid of a pest. However, it soon became apparent that mosquitoes were important vectors in France, and the presence of such a highly mobile and efficient vector provided an explanation for the movement of the disease to new areas (Fig. 9.7) and from wild to domestic rabbits. In the early days of the outbreak, there was often inadvertent transfer of disease between rabbit farms via infected rabbits still incubating the disease. Rabbit fleas are as common amongst wild rabbits in France as they are in Britain and undoubtedly contribute greatly to maintenance of enzootic myxomatosis throughout the year.

**Epidemiology**

With the knowledge that mosquitoes had been important vectors in Australia, it was...
soon shown that the prime suspect for transmission in the early epizootics in France, *Anopheles maculipennis atroparvus*, was an effective vector (Jacotot *et al.*, 1954). Subsequently, quiescent rabbit fleas were recovered from soil scrapings from deep burrows that had been abandoned by rabbits ten weeks earlier, following autumn epizootics of myxomatosis, and myxoma virus was recovered from these fleas (Joubert *et al.*, 1969). Arthur and Louzis (1988) postulated that eventually an enzootic–epizootic cycle involving reservoirs, vectors and susceptible rabbits developed. They suggested that the virus might overwinter in fleas in the soil of the burrow, in rabbits convalescing from infection, and in hibernating mosquitoes that might be contaminated with the virus. With the onset of warm weather, sharp mosquito-borne epizootics occurred among wild rabbits, rising to a peak in mid-summer and extending into autumn. These spread into domestic rabbits housed in non-mosquito-proof shelters.

Sightings of diseased rabbits fell when the wild rabbit population became greatly depleted in the mid-1950s, but after the attenuation of the virus and growing innate resistance in the rabbits in the late 1960s, the numbers of rabbits increased again. The seasonal and annual variations in myxomatosis then seen resulted from the interaction of the abundance of rabbits, their immune status (which was influenced by the age structure of the population) and the abundance of vectors. The occurrence of epizootics in southern France during summer and autumn was due to the concurrence of large numbers of aggressive mosquitoes and the presence of non-immune young rabbits. If there had been no myxomatosis the previous year, outbreaks often occurred in the spring and simulids and ceratopogonids were often involved (Joubert and Monnet, 1975). Spring outbreaks affected mainly young rabbits that had not been infected the previous summer, the resulting decrease in breeding females greatly depressing reproduction. Infection is maintained by rabbit fleas during autumn, winter and early spring, spread then being slow. Overwintering in the absence of obvious disease may be due to the persistence of virus on the mouth parts of fleas, which can remain in a quiescent state for some time in uninhabited burrows.

### Impact on wild rabbits

Myxomatosis clearly had a major impact on the number of wild rabbits that were available for hunting, and the bag did not reach even 20% of its pre-myxomatosis level until the 1961–62 season, although the recorded figures may partly reflect the paucity of hunters as well as of rabbits (Table 9.1). In the Sologne, a favoured département for rabbit hunting, the mean numbers shot per season (compared with

<table>
<thead>
<tr>
<th>Year</th>
<th>Number killed</th>
<th>Notes</th>
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<tbody>
<tr>
<td>1950–51</td>
<td>64,439,000</td>
<td></td>
</tr>
<tr>
<td>1951–52</td>
<td>37,723,000</td>
<td></td>
</tr>
<tr>
<td>1952–53</td>
<td>55,133,000</td>
<td></td>
</tr>
<tr>
<td>1953–54</td>
<td>6,721,000</td>
<td>of which 80% were from two unaffected hunts</td>
</tr>
<tr>
<td>1954–55</td>
<td>2,750,000</td>
<td>of which 87% were from one unaffected hunt</td>
</tr>
<tr>
<td>1955–56</td>
<td>2,404,000</td>
<td>of which 50% were from one hunt</td>
</tr>
<tr>
<td>1956–57</td>
<td>1,293,000</td>
<td></td>
</tr>
<tr>
<td>1957–58</td>
<td>3,357,000</td>
<td></td>
</tr>
<tr>
<td>1958–59</td>
<td>2,043,000</td>
<td></td>
</tr>
<tr>
<td>1959–60</td>
<td>4,074,000</td>
<td></td>
</tr>
<tr>
<td>1960–61</td>
<td>5,491,000</td>
<td></td>
</tr>
<tr>
<td>1961–62</td>
<td>10,906,000</td>
<td></td>
</tr>
</tbody>
</table>

aData from Giban (1956) and personal communication to Fenner (1963).
pre-myxomatosis figures) increased from 1.5% in 1953–58 to 13.5% in 1967–72 and 17.6% in 1972–77 (Arthur et al., 1980). However, after rabbits began to build up in numbers due to the presence of attenuated strains of virus and greater innate resistance, variations in the timing of major outbreaks of myxomatosis in relation to the hunting season caused large fluctuations in the numbers of rabbits shot. National surveys in France in 1977 and 1978 (Arthur et al., 1980) showed that rabbit density varied from place to place depending on environmental factors, such as type of soil and agricultural practice, and was also affected by hunting pressure and predation. At that time rabbits were most abundant in north-western France and much less common in the south and west, where in contrast to pre-myxomatosis times they often lived only in isolated pockets. Twenty years later (1997) the picture has not changed.

After the first disastrous epidemics, a variety of measures were undertaken by hunting organizations to mitigate the impact of myxomatosis. Vaccination with fibroma virus was carried out in an attempt to preserve some rabbits for shooting and hopefully to build up ‘barriers’ to the movement of myxomatosis. Later it was suggested that, as with domestic rabbits, wild rabbits should first be injected with fibroma virus followed 6–8 weeks later by SG33, both being administered by jet injector (Joubert et al., 1982; Fig. 9.8). Other early and unsuccessful methods for preserving rabbits as game animals included the introduction of cottontails (Sylvilagus floridanus), both as game animals and to hybridize with Oryctolagus, and a proposal to import rabbits from regions of Australia where innate resistance was high.

**Impact on domestic rabbits**

Myxomatosis was spread by mosquitoes from wild to domestic rabbits and initially it devastated the rabbit-breeding industry; it was estimated in 1954 that 30–40% of the industry had been destroyed. Vaccination afforded some protection, and with the reduction of the size of the wild rabbit reservoir of myxomatosis in the late 1950s new outbreaks in rabbitries became less common. Initially fibroma vaccine was used on a very large scale, amounting to tens of millions of doses annually; later it was used on a smaller scale, primarily to

**Fig. 9.8.** Louis Joubert (1922–1989). Born in Grenoble in 1922, Joubert enrolled in the National Veterinary School at Toulouse in 1939, graduating with honours in 1945 and becoming assistant to the Professor of Infectious Diseases. He continued his studies in veterinary medicine and in pharmacy, and in 1948 was appointed project director at the Veterinary School in Lyon, where he advanced to become a professor in 1962. His early research was focused on the epidemiology of zoonotic diseases, and after Jacotot had retired he became the leading expert on myxomatosis in France, showing particular interest in unusual insect vectors and especially in the ‘respiratory’ or ‘amyxomatous’ form of the disease. He was the senior author of a major book on myxomatosis, published in Paris in 1972–1973.
protect breeding animals. It was only moderately effective, and after limited trials with an attenuated myxoma virus vaccine developed in California (Saito et al., 1964), another attenuated vaccine (SG33) was developed in France (Saurat et al., 1978). Given to rabbits at 2–3 months of age, it was effective for about a year after vaccination. However, the use of the SG33 vaccine was found to have an immunosuppressive effect, which among rabbits housed under conditions of poor hygiene led to a variety of secondary bacterial infections (Brun et al., 1981; Godard, 1980). In such circumstances it was recommended that fibroma virus should be used for primary vaccination, followed by SG33 vaccine a month later.

The epidemiological cycle as seen in the 1980s differed according to the type of husbandry (Arthur and Louzis, 1988). Under conditions of traditional husbandry, in animal quarters with open contact with the outside world, the strains of virus were usually of the traditional nodular type, and were usually more lethal among the (genetically) unselected domestic rabbits than they were for wild rabbits. Outbreaks usually occurred in the autumn; there was a delay of 6–8 weeks between epizootic peaks in wild and domestic rabbits, the domestic rabbit peak coinciding with the arrival of mosquitoes within farm buildings (Puech, 1980). This form of myxomatosis could be prevented by efficient protection against the entry of mosquitoes by netting, unless animals harbouring the virus were brought in during the purchase of breeding animals.

Respiratory or non-myxomatous myxomatosis
Under intensive husbandry within closed mosquito-proof buildings, a non-myxomatous, pulmonary form of the disease, in which respiratory signs predominated, was most common (Brun et al., 1981a; see p. 216). It appeared to be transmitted by the inhalation of infective particles, both experimentally (Chantal, 1981) and in the rabbit farms. The incubation period varied between one and three weeks, and infection was manifested by swelling of the eyelids, genital and nasal lesions, lacrimation and a mucopurulent nasal discharge. This occurred primarily in premises in which there was poor ventilation and a high frequency of respiratory disease, and episodes of this form of myxomatosis were likely to occur at any time of the year. Most outbreaks occurred after recent introductions of rabbits from farms which had experienced infection during the previous 2–3 months. Such animals may have been incubating the disease for a longer period than usual, or, it has been suggested, they were asymptomatic carriers, in which reactivation occurred after the stress of transfer to new premises.

Changes in the Virulence of the Virus
Attenuated strains of myxoma virus were first recognized in 1955, in the département of Loiret (see Fig. 9.4B). Other strains with a similar degree of attenuation were soon found elsewhere in France (Fenner and Marshall, 1955; Siriez, 1960); the

Table 9.2. The virulence of field strains of myxoma virus in France in 1953, 1962, and 1968, calculated on the basis of mean survival times and expressed as percentages.a

<table>
<thead>
<tr>
<th>Year</th>
<th>Virulence grade</th>
<th>I</th>
<th>II</th>
<th>IIIA</th>
<th>IIIIB</th>
<th>IV</th>
<th>V</th>
</tr>
</thead>
<tbody>
<tr>
<td>1962</td>
<td>&gt;99</td>
<td>95–99</td>
<td>90–95</td>
<td>70–90</td>
<td>50–70</td>
<td>&lt;50</td>
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<td>1968</td>
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<td>19.3</td>
<td>34.6</td>
<td>20.8</td>
<td>13.5</td>
<td>0.8</td>
</tr>
</tbody>
</table>

aFrom Joubert et al. (1972), p. 132.

bFrom field observations.
virulence of these strains was not altered by several serial passages in the rabbit testis (Jacotot et al., 1956). Of ten French strains recovered in 1960–63, three were classified as of Grade IIIA virulence, three of Grade IIIB, three of Grade IV and one of grade V (Fenner and Ratcliffe, 1965). More recent data (Table 9.2) suggested that 15 years after the introduction of the virus a few highly virulent strains were still circulating, although the majority of strains were moderately or highly attenuated.

Endnotes

1Basser Library Archives, MS143/25/5A. Letters; G. Remaudière to F.N. Ratcliffe (22 January 1952), F. Fenner to Remaudière (15 February 1952) and reply (19 February 1952).

2Basser Library Archives, MS143/25/5A. Correspondence between G. Bouvier and Fenner about origins of ‘Lausanne’ strain, used to introduce myxomatosis into Europe.


4In a communication to Jacotot et al. (1954), M.E. Roubaud, an entomologist with special knowledge of mosquitoes, commented: ‘Au point de vue de la transmission de la myxomatose dans le milieu des clapiers, c’est cet anophèle A. maculipennis s. lat. qui apparait sans nul doute, en Europe, comme devant jouer le rôle principal. Dans les bois, dans les garennes, parmi les lapins sauvage, ce sont d’autres espèces culicidiennes qu’il convient d’incriminer, parmi lesquelles de nombreux Aedes, l’Anopheles claviger (bifurcatus) et surtout l’A. plumbeus’.

References


