

Unapparent foot and mouth disease infection (sub-clinical infections and carriers): implications for control

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Summary

Unlike animals which are carriers of foot and mouth disease (FMD), sub-clinically infected animals may be highly contagious. The implications of sub-clinical infections for the control of FMD are serious because such animals are likely to disseminate the disease when in contact with susceptible livestock. Recent dissemination of FMD virus (FMDV) in Europe shows that sub-clinically infected animals render trade in animals or animal products a potential risk for importing countries. This clearly demonstrates that the paradigm 'free of FMD without vaccination' is not synonymous with 'risk-free'. The risk of introduction of sub-clinical FMD into FMD-free countries may increase significantly, with the occurrence of large susceptible animal populations, changed agricultural practices, expansion of trade in live animals and animal movements, increased trade in animal products and greater mobility of people. Such changes in circumstances require that national and international authorities remain continuously vigilant to determine any altered risk for importation of FMD.

A few historical reports and some recent observations in southern Africa indicate the possibility of dissemination of FMD by bovine carriers into herds of susceptible cattle. These reports have greatly influenced FMD trade policies and thus, FMD control and eradication strategies. However, other field evidence does not support this claim and several controlled experiments were unable to show that carriers are able to initiate disease. When millions of cattle were systematically vaccinated with good quality vaccines, FMD disappeared in spite of a large sentinel population in the form of calves and unvaccinated sheep and pigs. A low number of carriers most likely persisted, but they did not hamper the eradication of the disease.

Vaccination policies and trade regulation must be based on risk assessments taking these factors into consideration.

Keywords

Carriers – Control measures – Foot and mouth disease – Sub-clinical infection – Trade.

Introduction

Animals infected with foot and mouth disease (FMD) virus (FMDV) usually show clinical signs one to four days after being exposed to the virus. However, some animals may not develop much fever or obvious lesions. Thus, some animals may

become infected and excrete the virus despite not developing clinical signs of the disease. Animals that develop clinical signs and recover can also remain persistently infected. Animals in which FMDV persists in the oesophageal-pharyngeal region for more than four weeks after infection are referred to as carriers (55, 59). Martin *et al.* (51) assigned the term 'carrier' only to animals that are able to disseminate infection. The term 'FMD

carrier' does not fit the definition in that sense, because apart from historical evidence, transmission of FMD by carriers other than African buffalo (*Syncerus caffer*) and cattle in southern Africa (65, 66), has never been convincingly demonstrated. The term 'carrier' will be used throughout this paper, but with the understanding that this does not imply that such animals are necessarily contagious.

Sub-clinical infection

Infection of animals with FMDV where that event is not followed by clinical disease may be a result of the following:

- species in which clinical signs and lesions either do not occur or are difficult to observe
- characteristics of the virus strain involved (17, 20)
- exposure of partially immune populations (i.e. populations with low levels of herd immunity following vaccination) to infection.

Henderson (41) observed that a high proportion (11%, n = 411) of susceptible cattle exposed by contact to a variety of strains did not develop FMD, indicating that some individuals are apparently refractory to development of clinical disease. However, strain differences were observed with regard to spreading power or invasiveness as measured by the frequency of non-reactors and the mean incubation period of the reactors.

Unlike carrier animals, acutely infected animals that do not develop clinical disease may be highly contagious and able to disseminate infection. Recent examples of the epidemiological importance of sub-clinical infections are the spread of FMD in the United Kingdom (UK) and the subsequent spread of the disease to other countries. Foot and mouth disease was confirmed in the UK in pigs showing clinical signs at an abattoir in late February 2001 (36). The strain concerned was identified as the pan-Asian lineage of type O. The date of introduction into the UK is unclear, but back-tracing of infection suggests that the primary cases of infection may have occurred in early February in swill-fed pigs at a location situated 350 km from the abattoir (36).

Although there has been considerable speculation that FMD was present in sheep in the UK before February, evidence for this is weak. Subsequent spread possibly occurred from the infected pig farm to sheep on nearby mixed livestock farms (36). Some of these sheep moved through several markets, including the busiest sheep market in Europe in the middle of February. This resulted in dissemination of the virus in widespread locations from the south of Scotland to the southwest of England, and via traded animals to Northern Ireland and France and, eventually, the Netherlands (42).

The Netherlands imported calves from Ireland in late February 2001. The following day, the shipment of calves was delivered

to several locations in the Netherlands. According to European Union legislation, the calves had to make a rest stop, which happened to be in a rest-station at Mayenne in the west of France, the place where the first outbreak of FMD on mainland Europe was diagnosed in the middle of March (42).

Goats on a farm in the centre of the Netherlands developed FMD a few days after the farm had received calves from Mayenne. Although no clinical signs of FMD were apparent, three of the five calves had antibodies suggesting that they had been infected. The diagnosis of FMD in goats was confirmed by laboratory tests five days later (42). Unfortunately, the calves were not tested to determine whether the FMDV was present in their throats. It can only be assumed at this point that the disease probably entered the Netherlands through sub-clinically infected fattening calves that spread the infection to goats housed adjacently.

An interesting reference to Greece, cited by Barnett and Cox (13) states that FMD infection might have occurred asymptotically in sheep between 1994 and 1995.

As illustrated by the above episodes in the UK, the Netherlands and possibly Greece, the implications of sub-clinical infection for the control of FMD are serious, demonstrating that no trade in animals or animal products can take place without the risk of importing FMD. Although the status 'country or zone free of FMD where vaccination is not practised' is the category most sought after by many regions and countries, this experience clearly demonstrates that the paradigm 'free of FMD without vaccination' is not synonymous with 'risk free'.

Foot and mouth disease could be introduced into a zone or country anywhere in the world by the introduction of sub-clinically infected animals. In addition, the risk of introducing FMD by either clinically or sub-clinically infected animals increases significantly with the following:

- the creation of large susceptible animal populations (e.g. through non-vaccination policies) and changed agricultural practices (e.g. intensification of livestock production and global trade of livestock)
- the persistence of endemically infected regions that endanger those susceptible populations
- expansion of trade in live animals and animal products
- greater mobility of people.

Periodical adjustments of existing policies based on risk assessments are needed. In this way, optimal effectiveness of national and international preventive measures can be achieved. Revised rules should not create unacceptable risks for trade partners but, at the same time, prevention and control of FMD should be conducted with the least disruption of the farming community or the rural economy.

Sub-clinical infection of sheep is of particular importance as demonstrated by the involvement of these animals in the FMD epidemic which occurred in the UK in 2001. This suggests that FMDV may have continued to circulate in flocks for some time without the appearance of clinical signs before finally disappearing. In North Africa and the Middle East, this applies equally to goats.

Strain characteristics

Bouma and Dekker infected calves with an O type virus isolated in the Netherlands (20). The animals developed some minor clinical signs after intranasal exposure that might not have been recognised by the farmer or diagnosed as FMD by a veterinarian. Calves in contact remained clinically normal and did not develop detectable antibody. These findings are consistent with field observations in the Netherlands and prove that sub-clinical infections of calves may be common with this strain of virus.

In the experience of the authors, the clinical and epidemiological characteristics of the type O virus prevalent in the Netherlands and the UK in 2001 showed similarities with strains used as modified live virus vaccines during the 1960s in South America. The virulence of these vaccine strains was reduced for cattle by serial passage in foreign hosts (21). Not all vaccinated cattle developed antibodies, even though the virus could later be recovered from such animals (10, 11). The sub-clinical infection of cattle caused by these strains was also sometimes transmitted to contact cattle, with or without subsequent development of antibody. It was not unusual for pigs to contract FMD on farms where cattle were vaccinated with modified live strains, although the cattle remained asymptomatic.

Beard and Mason established the genetic basis for the pig-specific pathogenicity of the O strain isolated from the 1997 FMD outbreak in Taipei China (17). The virus recovered from infected pigs was unable to infect bovine thyroid cell cultures or to cause typical disease in cattle following inoculation into the epithelium of the tongue. They found this virus to be a deletion mutant similar to that which occurred in the above-mentioned chicken embryo-adapted live virus vaccines strains used in South America (54). With regard to the possible origin of strains with a tendency to cause sub-clinical infection, there is speculation as to whether such strains could have arisen naturally or were man-made and escaped into the environment.

Persistent infection

Cattle

For more than 100 years, cattle that recovered from FMD were thought to be able to initiate outbreaks of the disease (33, 55, 58). This suspicion was raised because of outbreaks that occurred in countries or areas free of FMD after the

introduction of healthy convalescent cattle from regions where the disease had occurred.

An interesting observation was made in the UK after the serious 1922 to 1924 FMD epizootic (58). Given the extent of the epidemic, the traditional slaughter policy was partly abandoned and 105 infected farms were isolated, without slaughter. From these farms, eight months later, a convalescent bull and a heifer were sold to a district where no disease had been observed. After introduction of these animals into the new herd, FMD occurred and was attributed to these animals.

Furthermore, imported Brazilian zebu cattle were probably the source of an outbreak in Mexico in the late 1940s (10, 24). Finally, there is evidence of involvement of (non-vaccinated) persistently infected cattle in outbreaks caused by South African Territories (SAT) 2 strains in Zimbabwe (62, 65), but this may be a special case related to SAT type viruses, not documented for types O, A and C of FMDV.

In the late 1950s and early 1960s, studies showed that in countries with endemic FMD, virus could be isolated from the mucous and cellular material collected from the oro-pharyngeal region of as many as half of the cattle population that had recovered from FMD (19, 57). In general, this was found to be true for all seven FMD serotypes (63). However, depending on the virus strain, breed of cattle and local circumstances, figures may vary and individual cattle will show differences in duration and level of virus in probang samples.

Reported or suspected FMDV dissemination by carriers in countries having experienced FMD outbreaks mostly occurred prior to 1960, during the era before introduction of vaccination, i.e. when a high proportion of recovered animals were carriers. Later, the introduction of vaccination drastically reduced the morbidity rate and the amount of virus circulating in livestock populations. Anderson *et al.* found that the incidence of carriers in a vaccinated zone of Kenya was 0.49% as compared to 3.34% in a non-vaccinated area (3).

In the early 1960s, before the start of the systematic vaccination programmes in Brazil, virus-positive probang samples were commonly recovered from over 50% of the cattle (57). Two decades later, in 1983 and 1984, when vaccination programmes were intensified, probang sampling of several hundreds of cattle in endemic areas of Brazil resulted in only a very small number of positive samples (M. Obdeijn da Silva, personal communication). Similar results were obtained when throat swabs were collected from slaughter cattle for virus isolation (P. Augé de Mello and M. Obdeijn da Silva, personal communication).

In countries where FMD was eradicated by systematically vaccinating the cattle population, transmission of disease from carrier cattle to sentinel unvaccinated young cattle or to other susceptible species that had not been vaccinated (pigs, sheep,

goats) has never been observed. In countries in Europe and South America, where after a period of 'freedom of FMD', vaccination was discontinued, no cases of FMD could be linked to the existence of carriers.

Sutmoller and McVicar performed controlled experiments in the hope of showing that carriers could initiate disease (60). However, close contact exposure of susceptible animals (cattle and pigs) with carrier cattle did not result in disease transmission. Even under circumstances where the carrier cattle and the susceptible contacts were stressed in various ways, including administration of corticosteroids, transmission failed to occur. The feet of the contact pigs were traumatised and they were also fed swine *Ascaris* larvae in order to provide stress and a suitable entry site for the virus. Other authors have also reported on the failure of attempts to demonstrate FMD transmission from persistently infected cattle to susceptible cohorts (1, 16, 19, 25, 43, 63). Many more attempts at carrier transmission have probably been made and not reported because of negative findings. Thomson pointed out that carrier transmission could simply be an infrequent stochastic phenomenon (63).

Except for the observation by Hedger and Condy (40) that two carrier buffalo infected in-contact cattle after long-distance transportation, there is little evidence that stress induces or increases FMDV excretion. Attempts to mimic stress reactions with corticosteroid treatments have resulted in decreases in virus in probang samples (9). In fact, FMDV rapidly became undetectable in the carrier cattle following corticosteroid treatment. Furthermore, when super-infected with virulent infectious bovine rhinotracheitis (IBR) virus, cattle persistently infected with FMDV failed to transmit FMDV to susceptible cattle with which they were in contact (47).

Thus, considering historical evidence, field observations and experimental results, transmission of FMDV by carrier cattle is a rare event in the opinion of the authors.

Sheep and goats

Persistent infection in sheep and goats has been less extensively studied than in cattle. Flückiger pointed out the possible role of carriers in other species such as goats (32). In general, sheep and goats develop persistent infection less frequently, and for shorter periods than cattle, and the carrier state lasts from 1 to 5 months only (23). However, in some animals, the carrier state may last up to 12 months (48, 56). Unequivocal evidence of transmission from carrier sheep or goats has never been demonstrated under experimental situations or in the field (1, 16, 56). Therefore, the risk associated with the failure to detect such animals is negligible.

Pigs

Convalescent or vaccinated pigs have never been shown to be persistently infected using conventional tests. Furthermore,

such pigs have never been incriminated as a cause of outbreaks. This was challenged recently by Mezenzio *et al.* who reported the identification of viral ribonucleic acid (RNA) in the blood of recovered swine and fluctuations of virus neutralisation activity in the sera shortly after the appearance of viral RNA in the serum (52). This RNA was presumed to be in the form of complexes with high levels of antibody. However, further research is required to confirm these results and their epidemiological significance remains to be determined.

Camelids

Llamas (*Llama glama*) do not become FMDV carriers (27, 46). Most llamas exposed to FMDV remain uninfected. Those that become infected only show the presence of virus in the pharyngeal mucosae for up to 14 days. Recovered animals do not transmit virus to other susceptible species (27, 46).

African buffalo

Most free-living populations of African buffalo in southern Africa have high infection rates with SAT type FMDV (30). In the Kruger National Park in South Africa, rates of persistent infection of buffalo are as high as 60% (3, 38, 39). Individual animals may maintain the infection for periods of at least five years (26), but in most buffalo, the rates peak in the one to three year age group (39). Individual buffalo may be persistently infected with more than one type of FMDV in the pharyngeal region (3, 38). Carriers transmit the infection poorly to cohorts and to other susceptible species and then, only following prolonged and intimate contact (4, 37, 62, 63, 64). For instance, in Botswana a high percentage of buffalo carried FMDV but no clinical signs of disease in either the buffalo or other susceptible species with which they were in close contact were observed and there was complete absence of the disease in domestic stock over an eight-year period (39). However, good molecular, epidemiological and circumstantial evidence shows that two recent outbreaks of FMD in cattle in South Africa were caused by buffalo that escaped from the Kruger National Park (22, 67). The principal and largely successful strategy against FMD in southern Africa has, for many years, relied on the separation of cattle from buffalo by using fencing and other measures (61).

In addition, susceptible cattle kept in close association over a period of two and a half years with African buffalo carrying FMDV did not result in the transfer of infection from buffalo to cattle during this period, although infection did take place between the buffalo (25).

Conversely, in a different study, transmission of a plaque-purified SAT 2 FMDV occurred erratically from artificially infected African buffalo in captivity to susceptible buffalo and cattle in the same enclosure (66). In some instances, transmission occurred only after contact between persistently infected carriers and susceptible animals lasting a number of months (66). Interestingly, on this occasion as well as on that

reported by Dawe *et al.* (28), the cattle involved were cows, and male buffalo were present in the pens on both occasions. Most previous unsuccessful experiments designed to test transmission between buffalo and cattle did not involve bulls (35), and neither did transmission experiments between cattle (60).

Sexual transmission of the disease from carrier buffalo bulls to domestic cows was suggested by Bastos *et al.* (15). SAT type virus was isolated from both semen and from sheath washes from a naturally infected African buffalo. This was considered a persistent infection since the virus genotype had not recently been circulating in the buffalo herd. The virus in the sheath wash of the buffalo bull presumably originated from the mucosal epithelial tissues of the prepuce. It is peculiar that cattle herds, including bulls, seem to be involved in some of the historically reported incidents of carrier-associated outbreaks (33, 58). The possibility of sexual transmission by carrier animals is interesting to pursue, both in cattle and sheep, since this route would involve direct placement of potential infection onto mucosa with potential virus receptors or cells which might uptake virus-antibody complexes.

Limited, isolated groups of buffalo can maintain the virus circulating in the herds for many years (26). Given that few infected buffalo develop clinical signs, the dynamics of these infections are difficult to discern. However, various lines of evidence have led to the conclusion that most young buffalo become infected at two to six months of age, when maternal antibodies wane (63). By the time buffalo in endemic regions reach one year of age, most animals have high antibody levels to all three SAT virus types (63). In the acute stage of infection, young buffalo excrete these viruses in roughly the same quantities and by the same routes as do cattle and are therefore potentially highly infectious (35). Such 'childhood epizootics' (63) may keep the virus circulating in the buffalo herd and cause transmission of FMD from buffalo herds to other wildlife and domestic species (28, 29, 40, 63, 66).

The SAT type viruses have developed an intimate relationship with the African buffalo. Whether this relationship depends on the SAT type virus, on the African buffalo, or both, provides interesting speculation. It is also unknown whether the A, O and C serotypes would act like the SAT viruses in the African buffalo or whether the SAT viruses possess special characteristics that make them such 'good' carrier viruses in the buffalo in the sense that efficient transmission occurs in this species.

Other wildlife

Clinical and serological evidence clearly shows that several species of African antelope can become infected with FMD (2, 31, 38, 63). Viral persistence in antelope has only been reported in the kudu (*Tragelaphus streptoceros*) in which – after artificial infection – virus was detected for almost five months

(38). In the same investigation, two wildebeest (*Connochaetus taurinus*) carried SAT 1 virus for 45 days after infection, but this was not confirmed in a subsequent study (2). Foot and mouth disease virus persistence for up to 57 days was found in sable antelope (*Hippotragus niger*) (31). Experimental studies have failed to provide evidence of viral persistence in impala (*Aepyceros melampus*) (2, 38) which is the most frequently affected species in southern Africa (63). In the opinion of the authors, wildlife is much more likely to spread FMDV in the clinical or sub-clinical state than as carriers.

Forman *et al.* studied the FMD-carrier state in three species of deer in the UK (34). Foot and mouth disease virus was seldom recovered from the oro-pharynx from red deer (*Cervus elaphus*) and roe deer (*Capreolus capreolus*) beyond 14 days post-exposure. Fallow deer (*Dama dama*) carried the virus for a minimum of five weeks.

White-tailed deer (*Odocoileus virginianus*) in the United States of America (USA) carried FMDV regularly up to five weeks after exposure, but one deer showed virus in the oesophageal-pharyngeal fluid as long as eleven weeks post-exposure (49).

Persistent infection and foot and mouth disease control

Notions, such as 'vaccination masks the disease', 'vaccination keeps the disease amongst us', 'vaccination causes carriers,' or 'vaccination is not safe', that surround the vaccination and carrier issue today, originated some three decades ago when FMD control was based on mass vaccination campaigns using aqueous vaccines. However, at the time, the vaccines were of variable quality and were not always available when needed. Furthermore, there was limited control on their use and application. At the time, this resulted in low vaccination coverage and variable herd immunity. The policy served to maintain the epidemiological *status quo* and, at best, limited morbidity. This situation remains unchanged in large parts of the world.

In addition, a few outbreaks occurred in Europe that were associated either with poorly inactivated vaccine or with virus escapes from vaccine production plants (18). Furthermore, severe outbreaks in the west of France, mainly in pigs, seemed to be vaccine-related (44). However, in the Netherlands during the 40 years in which FMD vaccine was used (over 200 million doses), no such cases occurred. Current FMD vaccines are safe to use given the development of improved inactivants (12, 14) and proper safety testing and alone, they will not cause sub-clinical disease or carriers.

Vaccinated animals may become carriers when in contact with diseased livestock (50), but in the experience of the authors, vaccinated animals are unlikely to become carriers when exposed to the relatively small amounts of FMDV disseminated by fomites or people. Furthermore, vaccination suppresses the

amount of FMDV disseminated – released or discharged – in the environment by limiting the number of diseased animals in the population.

When millions of cattle were systematically vaccinated with good quality vaccines, FMD disappeared in spite of a large sentinel population in the form of calves and unvaccinated sheep and pigs. A low number of carriers probably persisted, but those did not hamper eradication of the disease. When the Netherlands, in 1957, and later, other countries in Europe such as France and Germany, eradicated FMD by general vaccination of all the cattle (no other species were vaccinated), there were no further outbreaks that could be attributed to the existence of carriers.

The countries of the southern cone of South America (Argentina, south of Brazil, Chile and Uruguay) were also able to eradicate FMD by strategic and systematic vaccination of the cattle population only, without the slaughter of infected animals. These countries had large unvaccinated sheep populations in direct contact with the vaccinated cattle. However, after vaccination of the cattle was discontinued, the countries remained free of FMD for several years, without any indication that carriers played a role in perpetuating FMD infection either in vaccinated or unvaccinated cattle or in the susceptible sheep population.

During 2001 there was, however, increasing concern about the existence of high-risk zones where sporadic disease occurred in the northern part of the southern cone of South America, accompanied by illegal incursions of livestock from such zones. The introduction of infected animals from these zones destabilised the FMD situation in most of the southern cone of South America, leading to the loss of the FMD-free status of Argentina, Uruguay and the southern states of Brazil. After the re-introduction of FMD, Argentina commenced strategic vaccination of the cattle population, with the exception of Patagonia. The last notified outbreak occurred in the Cordoba Province in January 2002. Uruguay also successfully eradicated the disease by strategic vaccination and livestock movement controls in a short period (8). The last case in the southern states of Brazil occurred in July 2001.

In the opinion of the authors, the introduction of FMD into previously FMD-free zones was caused by the movement of clinically or sub-clinically infected animals and not by bovine carriers. The outbreaks which occurred in South America in 2001, as well as the introduction and spread of the disease in Europe, demonstrate the great importance of active surveillance and the weakness of the present reporting system.

Sub-clinical and persistent infection and trade regulations

A response from the Institute for Animal Health/World Reference Laboratory for FMD to the Royal Society Inquiry

into infectious disease of livestock states: 'The carrier state is the chief regulatory barrier to the use of vaccination against FMD' (9). Indeed, the notions that vaccine may mask the infection or that carriers maintain FMDV in a vaccinated population (45) have resulted in international trade restrictions that impose a heavy penalty on the use of vaccine in the form of import/export restrictions of animals and animal products (5, 6, 7).

In cases where stamping-out and serological surveillance are applied following an outbreak in a previously 'FMD-free country without vaccination', the FMD-free status can be regained three months after the last case is observed. The waiting period is also three months after the last vaccinated animal is slaughtered when stamping-out, serological surveillance and emergency vaccination are applied (53). This strategy was used in the Netherlands to control the recent FMD outbreak, but at tremendous cost. For a total of only 27 infected farms, a quarter of a million mostly healthy animals were killed with considerable social and economic consequences.

In a country that is FMD-free, but practises vaccination, 12 months after stamping-out of the last case are required to regain FMD-free status; vaccination, without stamping-out, results in a 24-month waiting period after the last case (53).

Since there is no cut-off point for the carrier state, the authors believe that these differences in waiting periods are unjustified. In the opinion of the authors, the length of the waiting period should be determined by the ability and capacity of the Veterinary Service of the country concerned to demonstrate, to the satisfaction of the international animal health community, the absence of FMDV infection in the country. The sooner the country or region shows the absence of viral activity, the earlier normal trade can be resumed. For instance, this can be achieved by post-outbreak surveillance using tests for non-structural protein antibodies. This surveillance must be based on statistically sound population samples, keeping in mind the variability of the test sensitivity. Serological surveys carried out rapidly and efficiently would demonstrate to the international community the existence of well-organised Veterinary Services which take the eradication of FMD seriously. This approach is more flexible than the present extensive waiting periods and would allow ministries of agriculture and the livestock industry to select the options that produce the most desirable results with the least disruption of production systems and the lowest cost to the community.

The episodes in the UK and the Netherlands showed that because of sub-clinical infections, no trade in animals or animal products can occur without the risk of importing FMD. The risk of introduction of sub-clinical FMD into FMD-free countries can increase significantly, with the occurrence of large susceptible animal populations, changed agricultural practices, expansion of trade in live animals and animal movements, increased trade in animal products and greater mobility of

people. Such changes in circumstances require the continuous vigilance of national and international authorities in the determination of altered risk for importation of FMD.

Risk assessments could indicate where and what actions may be taken to minimise the risk. Trade restrictions for vaccinated cattle populations to reduce the import risks must also be based on risk assessments. These must consider the possibilities that

vaccination may mask the existence of disease, that there may be carriers in the population, that those carriers may pose a risk for FMD-free countries and that currently, vaccines and diagnostic tests are available to mitigate the risk.



L'infection asymptomatique de fièvre aphteuse (infections subcliniques et porteurs) : conséquences pour la prophylaxie

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Résumé

À la différence des animaux « porteurs » du virus de la fièvre aphteuse, les animaux ne présentant pas de signes cliniques apparents de cette maladie peuvent être extrêmement contagieux. Les infections subcliniques ont de graves implications pour la lutte contre la fièvre aphteuse car les animaux atteints sont susceptibles de répandre la maladie lorsqu'ils entrent en contact avec du bétail sensible. La récente propagation du virus de la fièvre aphteuse en Europe prouve que le commerce des animaux ou des produits d'origine animale constitue un risque potentiel pour les pays importateurs. Elle indique clairement que le paradigme « indemne de fièvre aphteuse où n'est pas pratiquée la vaccination » ne signifie pas nécessairement « dépourvu de risques ». Le risque d'introduction de fièvre aphteuse subclinique dans des pays indemnes peut être aggravé significativement par la présence de vastes populations d'animaux sensibles, la modification des pratiques agricoles, le développement du commerce des animaux vivants et des échanges d'animaux, l'accroissement du commerce des produits d'origine animale et la mobilité accrue des populations humaines. Face à cette évolution, il appartient aux autorités nationales et internationales de rester vigilantes pour apprécier toute évolution du risque de fièvre aphteuse à l'importation.

Quelques rapports historiques et des observations récentes en Afrique australe évoquent la possibilité d'une diffusion de la fièvre aphteuse par des bovins porteurs de virus au sein de troupeaux d'animaux sensibles. Ces rapports ont fortement influencé les politiques commerciales concernant la fièvre aphteuse et, par conséquent, les stratégies de prophylaxie et d'éradication de la maladie. Toutefois, d'autres indices recueillis sur le terrain vont à l'encontre de cette affirmation ; par ailleurs, aucune des expériences réalisées en conditions contrôlées n'a pu démontrer la capacité de ces porteurs de virus à déclencher la maladie. La vaccination systématique de millions de bovins avec des vaccins de qualité avait entraîné la disparition de la fièvre aphteuse, malgré une importante population sentinelle constituée de veaux, ainsi que d'ovins et de porcins non vaccinés. Parmi ces derniers, un certain nombre de porteurs de virus sont vraisemblablement passés entre les mailles du filet, sans pour autant compromettre l'éradication de la maladie.

Les politiques de vaccination et la réglementation du commerce doivent s'appuyer sur des évaluations des risques et prendre ces facteurs en considération.

Mots-clés

Échanges internationaux – Fièvre aphteuse – Infection subclinique – Mesures de prophylaxie – Porteurs de virus.



Consecuencias de los casos asintomáticos de fiebre aftosa (infecciones subclínicas y portadores) para el control de la enfermedad

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Resumen

A diferencia de los “portadores”, los animales con infección subclínica pueden ser extremadamente contagiosos. Las infecciones subclínicas suponen un gran problema para el control de la fiebre aftosa, pues esos animales pueden propagar muy fácilmente la enfermedad cuando entran en contacto con ganado susceptible. Como ponen de manifiesto los recientes episodios de dispersión del virus de la enfermedad por Europa, la presencia de animales con infección subclínica hace que el comercio de animales o productos de origen animal presente riesgos para los países importadores, lo que demuestra a las claras que el paradigma “libre de fiebre aftosa sin vacunación” no es sinónimo de “exento de riesgo”. El riesgo de que ejemplares con infección subclínica introduzcan la fiebre aftosa en países libres de la enfermedad puede aumentar sensiblemente cuando hay grandes poblaciones de animales susceptibles, cambian los métodos de trabajo agrícola, se intensifican el comercio y el movimiento de animales vivos o sus derivados y se acrece la movilidad de las personas. La constante evolución de esos parámetros exige de las autoridades nacionales e internacionales una continua vigilancia para detectar cualquier alteración de los niveles de riesgo de importación de fiebre aftosa.

De algunos informes históricos y una serie de observaciones recientes en el sur de África se desprende que los bovinos portadores pueden transmitir la enfermedad a rebaños de ganado vacuno susceptible. Dichos informes han tenido una gran influencia en las restricciones comerciales ligadas al riesgo de fiebre aftosa, y por consiguiente en las estrategias de control y erradicación de la enfermedad. Pero hay otros datos obtenidos sobre el terreno que no avalan esas suposiciones, y en varios experimentos controlados no pudo obtenerse prueba alguna de que los portadores sean capaces de inducir la enfermedad. Cuando se administraron sistemáticamente vacunas de buena calidad a millones de cabezas de ganado, la fiebre aftosa desapareció pese a la existencia de una gran población de animales testigo compuesta por terneros y ovinos y porcinos no vacunados. Aunque es muy probable que subsistieran unos pocos portadores, ello no impidió la erradicación de la enfermedad.

Las políticas de vacunación y las normas comerciales deben basarse en evaluaciones de riesgos que tengan en cuenta los factores aquí enumerados.

Palabras clave

Fiebre aftosa – Infección subclínica – Intercambios internacionales – Medidas de control – Portadores.



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