The effectiveness of routine serological surveillance: case study of the 1997 epidemic of classical swine fever in the Netherlands

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Summary
The authors describe the value of routine serological surveillance in detecting the introduction of classical swine fever virus into a disease-free population. The first investigation concerned the question of whether the epidemic of classical swine fever (CSF), which occurred from 1997 to 1998 in the Netherlands, could have been detected using the existing monitoring system for notifiable diseases. The investigation used data from the CSF epidemic of 1997/1998 and from the existing monitoring system. Secondly, the probability of detecting a case of CSF using routine serological surveillance was modelled both for multiplier herds and for finishing herds, and then for different herd size categories. The first investigation concluded that the probability of detecting the epidemic at the current level of routine serological surveillance is very low. The second investigation concluded that even employing a sampling scheme of sixty blood samples per month, the probability of detecting an outbreak of CSF within forty days of the introduction of the virus, is less than 40%.

Keywords

Introduction
After the introduction of classical swine fever (CSF) virus into a country, some time will elapse before the disease is detected. The high risk period (HRP) defines the period in which the virus can spread freely to other herds (5). This HRP begins when the first animal is infected and ends when all eradication measures are in full operation. The length of the HRP is one of the most important parameters in determining the magnitude of an epidemic. The HRP can be divided into two periods. The HRP\textsubscript{1} is defined as the period between introduction of CSF virus into a region and the detection of the first outbreak. The HRP\textsubscript{2} is defined as the period between detection of the first outbreak and the time when all measures considered have come into effect. The probability of spread of CSF virus to other herds during the HRP depends primarily on the length of the HRP, the spread of CSF virus within herds and the rate and nature of contacts between infected herds and non-infected herds. The length of the HRP\textsubscript{1} depends largely on the alertness, skill and motivation of farmers, veterinary practitioners and post-mortem laboratories, but also on the virulence of the CSF virus strain (3). The length of the HRP\textsubscript{2} in recent epidemics of CSF in Western Europe has varied...
from three weeks to nine weeks. In 60% of the epidemics, the HRP1 was five weeks or more (3, 13). In the 1997-1998 epidemic of CSF in the Netherlands, the first infected herd was detected on 4 February 1997, approximately six weeks after infection. An additional thirty-five herds were estimated to have become infected during the HRP1, of which, fourteen herds were due to trade on 4 February 1997 or on the previous day (11). Thus, for this epidemic, the routine measures taken to detect the disease at an early stage, such as clinical inspection, post-mortem examination and examination of tonsils, did not prove to be effective enough to detect the infection at an early stage (3).

Since December 1993, a national monitoring programme for swine vesicular disease (SVD) virus and later for other notifiable diseases, called the herd disease control regulation (HDC regulation), has been utilised in the Netherlands (6). The HDC regulation consists of a four-monthly clinical inspection and serological examination of all swine herds in the Netherlands. The existence of the HDC regulation led to the (political) question of whether the epidemic of CSF in the Netherlands could have been detected earlier if the available blood samples had also been tested for antibodies to CSF virus.

In the present study, the authors examine the question of whether routine serological surveillance is a useful tool for the reduction of the HRP1 of CSF. Firstly, the authors investigate the probability that the epidemic of CSF in the Netherlands in 1997/1998 would have been detected earlier by use of the blood samples collected within the framework of the HDC regulation. Secondly, in a modelling study, the usefulness of serological surveillance for early warning of CSF is examined in a more general context.

### Materials and methods

#### The herd disease control regulation

Within the framework of the HDC regulation, all swine herds (except herds of less than five animals) are clinically inspected by a veterinarian every four months. In addition, blood samples are collected and tested for antibodies to SVD virus. In multiplier herds, the number of samples taken is proportional to herd size based on the sows present. In fattening or rearing herds, samples are taken in proportion to herd size and in as many compartments as possible, with an optimum of one pig per pen. In mixed herds, only sows are sampled. The herd sampling was based on a hypergeometric distribution and a 95% probability of finding at least one seropositive animal if 25% or more of the animals in the herd are seropositive for SVD (6). Table I shows the number of samples collected in the framework of this regulation for different herd size categories.

### Data from the 1997-1998 epidemic in the Netherlands

Data were collected from herds that most likely became infected before 4 February 1997, the day the first CSF outbreak was detected. The estimated date of infection and the supposed infection source were derived from the interview reports of the National Inspection Service for Livestock and Meat (RVV). A file from the reference laboratory of the Institute for Animal Science and Health (ID-DLO) in Lelystad, describing laboratory results was also available. Numbers of animals per farm, herd type and last date of clinical inspection and blood sampling performed under the HDC regulation were derived from the HDC regulation database. In this study three different herd types were distinguished, as follows:

- multiplier herds (producers of breeding stock or finishing piglets)
- finishing herds (producers of fatteners)
- mixed herds (a combination of multiplier and finishing herds).

Because of the similarity between finishing and rearing herds in terms of production systems and housing, rearing herds (rearing of replacement gilts) were treated as finishing herds.

#### Serological tests

Serum derived from all blood samples collected before depopulation was tested for antibodies against CSF virus in an enzyme-linked immunosorbent assay (ELISA) (1). To ensure that the serological testing was CSF-specific, samples that twice showed an inhibition of 30% or over were retested in the neutralising peroxidase-linked assay (14), using CSF strain Brescia and border disease virus strain F and/or bovine virus diarrhoea virus strain Oregon. The time from collection of the sample until notification of the test results was approximately six days. The probability that a pig is seropositive, as a function of time $t$, since infection with the combination of the two serological tests, has been described as (12):

$$P(\text{detecting individual pig } p_{i}(t)) = \frac{0.99374}{1 + e^{-0.4800(t - 18.4512)}}$$

### Table I

<table>
<thead>
<tr>
<th>Herd size</th>
<th>Number of samples</th>
</tr>
</thead>
<tbody>
<tr>
<td>1-10</td>
<td>All pigs (to a maximum of 7)</td>
</tr>
<tr>
<td>11-30</td>
<td>9</td>
</tr>
<tr>
<td>31-120</td>
<td>10</td>
</tr>
<tr>
<td>&gt; 120</td>
<td>12</td>
</tr>
</tbody>
</table>
A graphic reproduction of this function is shown in Figure 1. The specificity of the combined tests was assumed to be 1.

![Graph showing time-dependent cumulative probability curve for detection of a seropositive pig using combined serological tests]

**Analysis of data**

The equations used for different processes are explained below.

An epidemic was denoted as detected if at least one infected herd was detected. The probability of detecting the epidemic depended on the herd-level sensitivity (HSE) of each herd, which became infected during the HRP1, and could be estimated as:

\[ P(\text{detecting epidemic}) = 1 - \Pi (1 - \text{HSE}) \]

where:

\[ P(\text{detecting epidemic}) = \text{the probability of detecting the epidemic,} \]

\[ \text{HSE} = \text{herd-level sensitivity (the probability of a truly infected herd being classified as infected by a test based on a random sample of animals) (9),} \]

\[ (1 - \text{HSE}) = \text{the probability that each herd, is not detected.} \]

A herd was denoted as positive if at least one pig was tested positive. The equation for herd-level sensitivity is as follows (9):

\[ \text{HSE} = 1 - (1 - \text{SP})^n \]

where:

\[ \text{HSE} = \text{herd-level sensitivity,} \]

\[ \text{SP} = \text{the seroprevalence (the proportion of seropositive pigs in a herd), and} \]

\[ n = \text{number of blood samples.} \]

The transmission of the virus in a herd could be described by a stochastic SIR model, in which individuals in a population are classified as either susceptible, infectious or removed (2). At the beginning of infection when the number of infectious pigs is small compared to the number of susceptible pigs, and under the assumption of random mixing of pigs in a herd with only one type of infectious or susceptible pig (sows, fatteners or replacement sows), the number of seropositive pigs (S) at day \( t \), could be estimated as follows (adapted from 10):

\[ S(t) = I_0 e^{r(t-18.5)} \]

where:

\[ S(t) = \text{number of seropositive pigs at day} \ t, \]

\[ t = \text{number of days since introduction of the virus,} \]

\[ I_0 = \text{number of infected pigs at} \ t = 0, \text{and} \]

\[ r = \text{the growth rate parameter.} \]

The growth rate parameter \( r \) could be estimated as follows (10):

\[ r = \frac{\ln (R_0)}{T_g} \]

where:

\[ r = \text{the growth rate parameter,} \]

\[ R_0 = \text{the reproduction ratio defined as the mean number of individuals infected by one infectious individual, and} \]

\[ T_g = \text{the mean generation interval (average time between infection of an individual and infection of the individuals that were infected by this individual) (12).} \]

**Analysis of the probability of detecting the 1997-1998 epidemic of classical swine fever using blood samples collected within the framework of the herd disease control regulation**

The growth rate parameter of each individual herd that became infected during the HRP1 was estimated by substituting the observed seroprevalence at depopulation and the time between infection and depopulation into equation [4], assuming one introduction at \( t = 0 \). Then, by
replacing the time between infection and depopulation in equation [4] with the time between infection and HDC sampling, and using the previous estimated growth rate parameter, the seroprevalence at HDC sampling for that particular herd was estimated. Equation [3] was then used to estimate the herd-level sensitivity for each individual herd on the day of HDC sampling. Finally, equation [2] was used to estimate the probability of detecting the epidemic by testing the HDC samples for antibodies to CSF virus.

Modelling the herd-level sensitivity for detecting classical swine fever by serological surveillance

For regular routine surveillance, the probability of a herd being first visited at day t after infection is independent of the date of virus introduction and has a uniform distribution as follows:

\[
P(\text{visit}_{t}) = \left( \frac{1}{\Delta T} \right)
\]

where:

\[
P(\text{visit}_{t}) = \text{the probability of a herd being visited at day t after infection, and}
\]

\[
\Delta T = \text{the average time (in days) between two HDC visits.}
\]

The herd-level sensitivities for different sampling schemes were modelled for the first sixty days post infection according to the sampling schemes given in Table II.

Table II
Blood sampling schemes used for various sizes and types of herds

<table>
<thead>
<tr>
<th>Blood sampling</th>
<th>Multiplier herds</th>
<th>Finishing herds</th>
</tr>
</thead>
<tbody>
<tr>
<td>12 samples each month</td>
<td>× × × × ×</td>
<td>× × × × ×</td>
</tr>
<tr>
<td>60 samples each month</td>
<td>× × × × ×</td>
<td>× × × × ×</td>
</tr>
<tr>
<td>12 samples every 4 months</td>
<td>× ×</td>
<td>× ×</td>
</tr>
<tr>
<td>60 samples every 4 months</td>
<td>×</td>
<td>×</td>
</tr>
</tbody>
</table>

With a four monthly sampling scheme, the probability that the herd would be found to be positive if tested on or before t days post infection is as follows:

\[
Q(t) = \left( \sum_{i=1}^{\frac{t}{185}} \frac{P_{i}}{121} \right)
\]

where:

\[
Q(t) = \text{the probability that the herd was detected on or before t days post infection, and}
\]

\[
P_{i} = \text{the probability that the herd was found to be positive if tested on day i post infection.}
\]

With a monthly sampling scheme, a herd would be visited twice over a period of sixty days, assuming a second visit thirty days after the first visit. For the first thirty days, the probability that the herd would be found positive on or before t days post infection could be calculated using equation [7], replacing 121 by 30. However, for the next thirty days, this probability also depends on the probability of not detecting the herd at the first visit:

\[
Q(t) = Q(30) + \left( \sum_{i=31}^{60} \left( 1 - P_{i-30} \right) \frac{P_{i}}{30} \right)
\]

for 31 \leq t \leq 60

Laevens et al. estimated \( R_{0} \) for finishing herds to be 13.5 (8). Stegeman et al. estimated \( R_{0} \) for multiplier herds to be 2.9 and \( T_{S} \) in general to be 10 (12). Furthermore, \( I_{0} \) was always assumed to be 1. In consequence, equation [4] became either:

\[
S(t) = e^{0.106(t - 18.5)}
\]

for multiplier herds, or

\[
S(t) = e^{0.260(t - 18.5)}
\]

for finishing herds.

The number of seropositive pigs per day post infection was estimated by using these equations. For each sampling scheme the herd-level sensitivity at day t post infection was calculated by substituting the corresponding seroprevalence and sample size in equation [3]. Finally, the cumulative probability of detecting a positive herd on or before t days post infection was calculated by using equation [7] for each four monthly sampling scheme and equation [8] for each monthly sampling scheme.

Results

Probability of detecting the 1997-1998 epidemic of classical swine fever using the blood samples collected within the framework of the herd disease control regulation

In the epidemic of 1997-1998 in the Netherlands, twenty-one herds became infected with CSF virus before 4 February 1997. Within this group, the estimated time until detection varied from fifteen days to forty-five days. Before the infected herds were depopulated, blood samples were collected from fifteen herds and examined for antibodies against CSF virus (Table III). Table III also illustrates the influence of time on the degree of seroprevalence.
Table III
Estimated time until detection and estimated seroprevalence at depopulation of herds which became infected with classical swine fever virus within the first of the high risk periods (HRP1) of the 1997-1998 epidemic of classical swine fever in the Netherlands

<table>
<thead>
<tr>
<th>Estimated time until detection</th>
<th>Number of herds not tested</th>
<th>Number of herds tested</th>
<th>Estimated seroprevalence and sample size (in brackets) for herds with seroprevalence &gt; 0</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 20 days</td>
<td>1</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>20-24 days</td>
<td>4</td>
<td>7</td>
<td>0.02 (111); 0.03 (30); 0.04 (113)</td>
</tr>
<tr>
<td>25-29 days</td>
<td>1</td>
<td>2</td>
<td>0.01 (145); 0.03 (362)</td>
</tr>
<tr>
<td>≥ 30 days</td>
<td>0</td>
<td>4</td>
<td>0.11 (117); 0.13 (175); 0.17 (60); 0.34 (50)</td>
</tr>
<tr>
<td>Total number of herds</td>
<td>6</td>
<td>15</td>
<td></td>
</tr>
</tbody>
</table>

Blood samples were collected from five herds within the HRP1 for the HDC regulation. From four of these five herds, the HDC blood samples were collected within ten days of the estimated date of infection (Table IV). Given a time lag of 18.5 days to allow for the formation of antibodies, the probability of the presence of a seropositive pig in a herd within ten days of infection can be ignored (equation [4]).

The combined probability of detecting the epidemic, based on the herd-level sensitivity of the five herds which were sampled within the HRP1, was estimated to be 0.39. This probability depended entirely on the contribution of herd 'A'.

Even if the herd-level sensitivity of herd 'A' had been 100% on the day of the HDC sampling, detection of the epidemic would have occurred, at the most, only two days earlier. This is calculated by taking the time between blood sampling of herd 'A' at the HDC visit and depopulation, minus the time needed (approximately six days) for notification of the test results (Table IV).

Modelling the probability of detecting an epidemic of classical swine fever at an early stage by serological surveillance

The cumulative probability functions for detecting an infected herd within t days post infection, for the different sampling schemes, are illustrated in Figure 2 for multiplier herds and in Figure 3 for finishing herds.

In the first part of the curves, the cumulative probability of detecting an infected herd is influenced by the herd-level sensitivity. When the herd-level sensitivity becomes one, this cumulative probability depends only on the timing of the sampling (number of days post infection).

Taking into account the time needed for notification of the test results (approximately six days), the cumulative probabilities of detecting an infected herd within forty days post infection was less than 0.40 for all sampling schemes. With a sample size of sixty blood samples every four months, these cumulative probabilities were still beneath 0.25 after sixty days.

Discussion

Probability of detecting the 1997-1998 epidemic of classical swine fever using the blood samples collected within the framework of the herd disease control regulation

The probability of detecting the 1997-1998 epidemic of CSF in the Netherlands using the blood samples collected within the framework of the HDC regulation was estimated to be 0.39. Although this probability depended entirely on the probability of detecting herd 'A', detection of the epidemic could have occurred no more than two days earlier. On
Fig. 2
Time-dependent cumulative probability functions for detection of an infected multiplier herd during the first sixty days post infection, using serological surveillance

The function is illustrated in respect to herd-level sensitivity and herd and sample size categories with a monthly or a four-monthly sampling scheme and assuming a reproduction ratio ($R_0$) of 2.9. The curves cover the first sixty days post infection and are based on two visits in a monthly scheme with a period of thirty days between the two visits, and on one visit in a four-monthly scheme.

5 February, the day after detection of the epidemic, the twelve HDC blood samples of herd 'A', collected on 27 January 1997, were tested for antibodies to CSF virus, and all tested negative. Consequently, the conclusion reached by the authors, that the epidemic would not have been detected by serological screening if the HDC blood samples were examined for antibodies directed against CSF virus, is justified.

Data concerning the first outbreaks were not always available or complete. The date used as the most likely infection date was that stated by the department of epidemiology of the disease control centre. However, these were often estimations from a range of possible infection dates. In the hectic first days after detection of the epidemic, six herds were not serologically examined at depopulation. These six herds also received no visit under the HDC regulation within the HRP, which means that these herds could not possibly have contributed to the probability of detecting the epidemic.

With a herd-level sensitivity of 0.39 (herd 'A'), the probability of not detecting a truly infected herd is 0.61. The results of examination of the blood samples of herd 'A' were in accordance with the estimated herd-level sensitivity on the day of the HDC visit.

Modelling the probability of detecting classical swine fever at an early stage by serological surveillance

The cumulative probabilities of detecting an infected herd by routine serological surveillance within forty days of the introduction of the virus into a herd, appeared to be beneath 0.40 for all tested sampling schemes, even if a monthly sampling scheme with sixty blood samples was accomplished. Therefore, routine serological surveillance as an early detection system for classical swine fever does not appear appropriate.

The most critical reason for this failure is the low level of infected pigs at the early stage of an outbreak. Furthermore, a number of days must pass before the level of antibodies is at a sufficiently high level to be reliably detected by the serological tests (equation [1]). Effectively, equation [4] is the number of infected pigs, with a time lag of 18.5 days to allow for the...
The number of infected pigs at day $t$ after introduction of the virus into the herd depends on the number of introductions, $I_0$, at $t = 0$, the reproduction ratio, $R_0$, and the generation interval of ten days (equations [4] and [5]).

The value of $I_0$ depends on how the virus is introduced into a herd. $I_0$ could correspond to a large proportion of the herd if the cause of the infection was, for example, swill feeding, in which many pigs might be infected simultaneously. This would increase the probability of early detection by serological surveillance because the number of seropositive animals from the eighteenth day would be fairly large. However, swill feeding is illegal in the Netherlands.

The generation interval was estimated from the CSF epidemic in 1997-1998, to be equal to ten days (12). The reproduction ratio, $R_0$, for multiplier herds was also estimated from that epidemic, which was caused by the CSF Paderborn strain (12). The reproduction ratio, $R_0$, for finishing herds was estimated from an experimental infection of finishing pigs with the CSF Lorraine strain (8). Both strains are believed to be moderately virulent strains. In an epidemic of CSF caused by a CSF strain of low virulence, clinical signs of the infection will be less obvious. In this case, serological testing of blood samples could be helpful. If serological evidence can be observed in advance of clinical signs then routine serological surveillance should provide an effective method of detecting the epidemic. However, based on the results of the current study, routine serological surveillance could never lead to a short HRP.$^1$

For the twelve blood samples of the HDC regulation, a seroprevalence of 0.25 could be detected with a 95% probability. Because the purpose of this study was to
investigate the probabilities of earlier detection of an infected herd through serological surveillance, a sample size of sixty blood samples was also investigated. Using sixty blood samples, a seroprevalence of 0.05 could be detected with a 95% probability (9).

In estimating the probabilities of detecting CSF by serological surveillance, the herds that were sampled were assumed to be homogeneous. This means that only one type of susceptible pig and only one type of infectious pig were present in the herd. The authors also assumed that these types of pig could only be sows or finishing pigs. The presence of suckling or weaned piglets in multiplier herds was not considered. Laevens et al. estimated a reproduction ratio for weaned pigs of $R_0 = 81.3$ (7). The presence of infected piglets in a multiplier herd could lead to multiple introductions in the sampled sow population and would therefore probably lead to a faster spread of the infection. This same argument could be used for the presence of finishing pigs in mixed herds. The inclusion of suckling and weaned piglets and, if present, of finishing pigs in the sampling scheme of (mixed) multiplier herds would probably improve the probability of detecting an infected multiplier herd by serological surveillance. However, Van Nes stated that the very complex models needed in order to cope with the presence of many different types of pigs make analytical solutions complex or even impossible (15).

The assumption of random mixing made in this part of the study could be invalidated by the housing system. The housing system restricts the number of different direct contacts that pigs can have with each other. In the Netherlands this is of special importance for finishing herds. However, due to indirect contacts via the farmer, an approximation of random mixing is possibly established. This assumption leads to an overestimation of the within-herd $R_0$ and consequently to an overestimation of the probability of detecting an infected herd. Therefore, the estimated probabilities must be seen as best-case probabilities, because of the necessary assumptions.

The reproduction ratios that were used for finishing herds in this model were estimates from a single experiment and a single epidemic, both with a moderately virulent virus strain. According to the reported high-risk periods of recent epidemics of CSF in Europe, most of the epidemics seemed to be caused by moderately virulent virus strains. Therefore, for most epidemics, the conclusion that routine serological surveillance is inappropriate as an early detecting system for CSF seems to be justified.

**General discussion**

The general conclusion of this study is that routine serological surveillance would not have contributed to the detection of the 1997-1998 CSF epidemic in the Netherlands, and would probably have little value in detecting an outbreak of CSF. However, these conclusions concern the detection of a first outbreak. The value of routine serological screening is to confirm that no unnoticed outbreaks have occurred. Although important, this application of serological surveillance is beyond the scope of this paper and will not be further discussed.

The two most important shortcomings of routine serological surveillance are the fact that the date of sampling is independent of the date of infection and the randomness of the sampling within a herd. Serological surveillance could be valuable if the time and location of introduction of the virus into the herd was known beforehand. This is clearly in contradiction with the purpose of routine serological surveillance. However, experts estimated the relative importance of virus introduction into the Netherlands to be almost 60% for import of livestock and almost 14% for returning livestock trucks from abroad (4). Serological surveillance could be used far more specifically by intensive screening of herds with contacts abroad and with respect to the time and type of those contacts. Even in this case, serological surveillance has limits in consequence of the low levels of seropositive animals present in the first weeks of the outbreak and the time-dependent probability of the serological tests to detect a seropositive pig.

Sampling at a higher frequency than once a month is not a feasible improvement because this would produce an immense amount of blood samples. With 20,000 pig herds in the Netherlands, even a sampling scheme of twelve blood samples collected every month would result in collecting and testing more than 12,000 blood samples every working day.

Alertness for clinical symptoms of an infectious disease is still the most important method for timely identification of an outbreak of CSF. If atypical signs of an infectious disease or unexplained sudden deaths occur in a herd, complementary measures should be established immediately. For example, if medication obtains no results within three days of administration, blood samples from pigs with disease signs could be collected for virus isolation at the laboratory. Sending a number of pigs with disease signs to a laboratory for post-mortem investigation could also be helpful in timely identification of an outbreak of CSF. When unexplained sudden deaths occur in a herd, the affected animals should always be sent to the laboratory for post-mortem investigation. Therefore, more emphasis should be placed on improving the skills of farmers and practitioners in recognising the clinical symptoms of CSF. This could be achieved through training and publications in specialist magazines.

**Conclusions**

Routine serological screening for antibodies against CSF virus as part of the HDC regulation would not have resulted in the

Routine serological surveillance is, in general, not an appropriate instrument to reduce the HRP₁ of an epidemic of CSF.

A more targeted sampling scheme could improve the opportunities for detection of outbreaks by serological surveillance, but will never lead to a substantially shorter HRP₁.

The real value of serological surveillance is to confirm that no unnoticed outbreaks have occurred.

Alertness for clinical symptoms of an infectious disease, accompanied by pathological and virological examination if atypical signs of an infectious disease occur, is still the most effective method for timely identification of an outbreak of CSF.

More emphasis should be placed on improving the skills of farmers and practitioners in recognising clinical symptoms of CSF.

Acknowledgements
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L'efficacité de la surveillance sérologique de routine : l'exemple de l'épizootie de peste porcine classique survenue en 1997 aux Pays-Bas


Résumé
Les auteurs évaluent l'efficacité de la surveillance sérologique de routine pour détecter l'apparition du virus de la peste porcine classique dans une population animale indemne. Une première enquête a tenté de déterminer si le système de suivi épidémiologique des maladies à déclaration obligatoire actuellement en vigueur aurait pu détecter à temps l'épizootie de peste porcine classique qui a frappé les Pays-Bas en 1997-1998. Cette enquête a utilisé les informations relatives à l'épizootie de 1997-1998 et au système de surveillance actuel. Dans un deuxième temps, un modèle a été conçu pour déterminer les probabilités que la surveillance sérologique de routine détecte l'apparition d'un cas de peste porcine classique dans un élevage de sujets reproducteurs, dans un élevage de porcs charcutiers, ainsi que dans des élevages de différentes tailles. La première enquête souligne la très faible probabilité que la surveillance sérologique de routine pratiquée actuellement puisse détecter une épizootie. D'après les résultats de la seconde enquête, la probabilité de détection d'un foyer de peste porcine classique dans les quarante jours suivant l'introduction du virus serait inférieure à 40 %, même en analysant 60 prélèvements de sang chaque mois.

Mots-clés
Détection précoce – Pays-Bas – Période à haut risque – Peste porcine classique – Surveillance sérologique de routine.
Eficacia de la vigilancia serológica sistemática: el caso de la epizootia de peste porcina clásica de 1997 en los Países Bajos


Resumen
Los autores evalúan la eficacia de la vigilancia serológica sistemática para detectar la penetración del virus de la peste porcina clásica en una población libre de la enfermedad. En una primera investigación se trató de determinar en qué medida la epizootia de peste porcina clásica que sufrieron los Países Bajos entre 1997 y 1998 podía haberse detectado mediante el sistema vigente de monitoreo de enfermedades de declaración obligatoria. Para ello se utilizaron los datos de aquella epidemia y del sistema de monitoreo. Después se construyó un modelo para estimar la probabilidad de detectar un caso de peste porcina clásica mediante procedimientos de vigilancia serológica sistemática, en rebaños de multiplicación como de engorde, y en rebaños de distintos tamaños. De la primera investigación se infirió que, con el nivel actual de vigilancia serológica, la probabilidad de detectar la epizootia era muy baja. La segunda investigación concluyó que, aun utilizando un programa de muestreo de sesenta muestras sanguíneas mensuales, la probabilidad de detectar un brote de peste porcina clásica en los cuarenta días siguientes a la introducción del virus era inferior a un 40%.

Palabras clave
Detección precoz – Países Bajos – Período de alto riesgo – Peste porcina clásica – Vigilancia serológica sistemática.

References


